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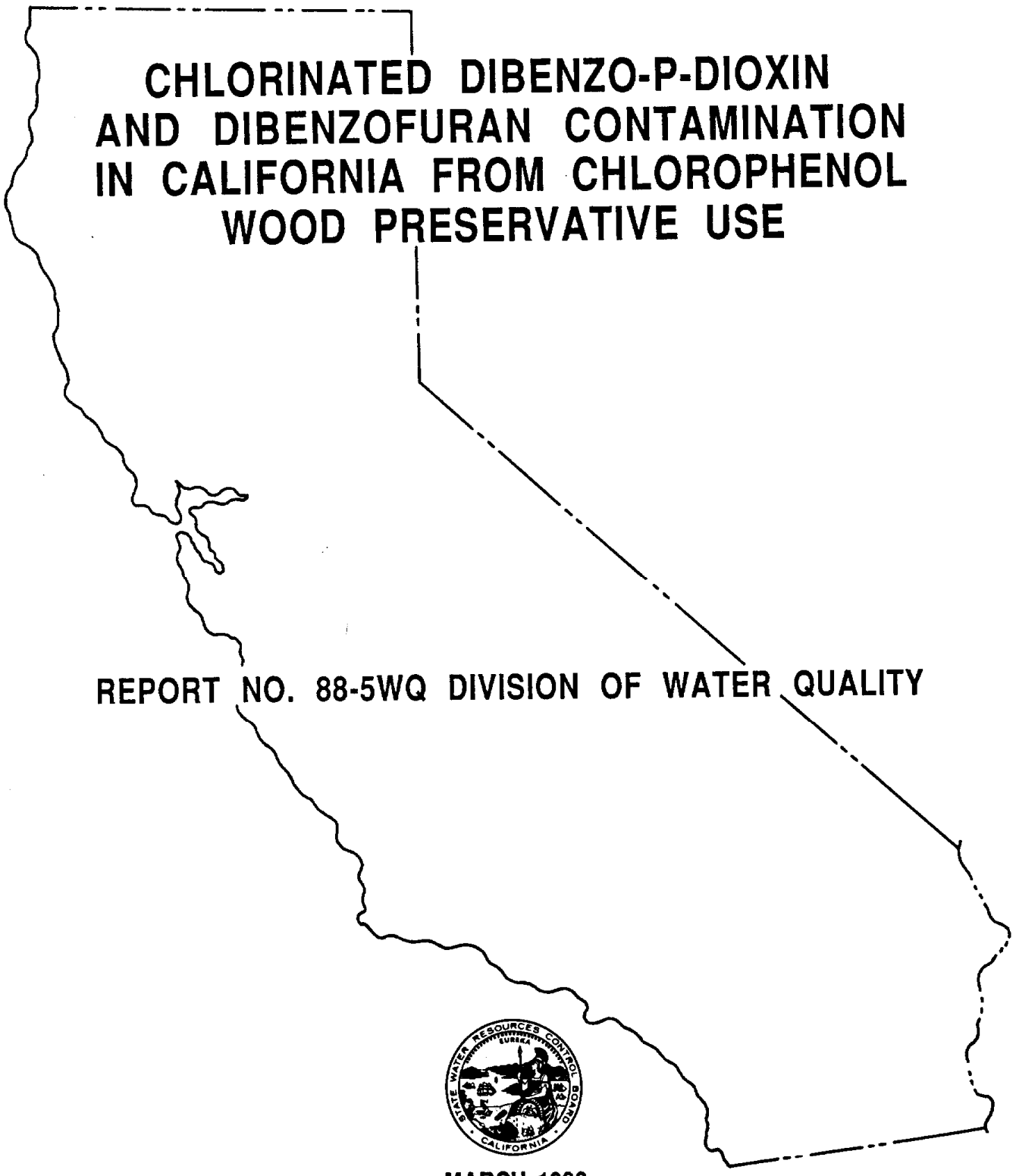

REPORT NO. 88-5WQ DIVISION OF WATER QUALITY

MARCH 1988

STATE WATER RESOURCES CONTROL BOARD

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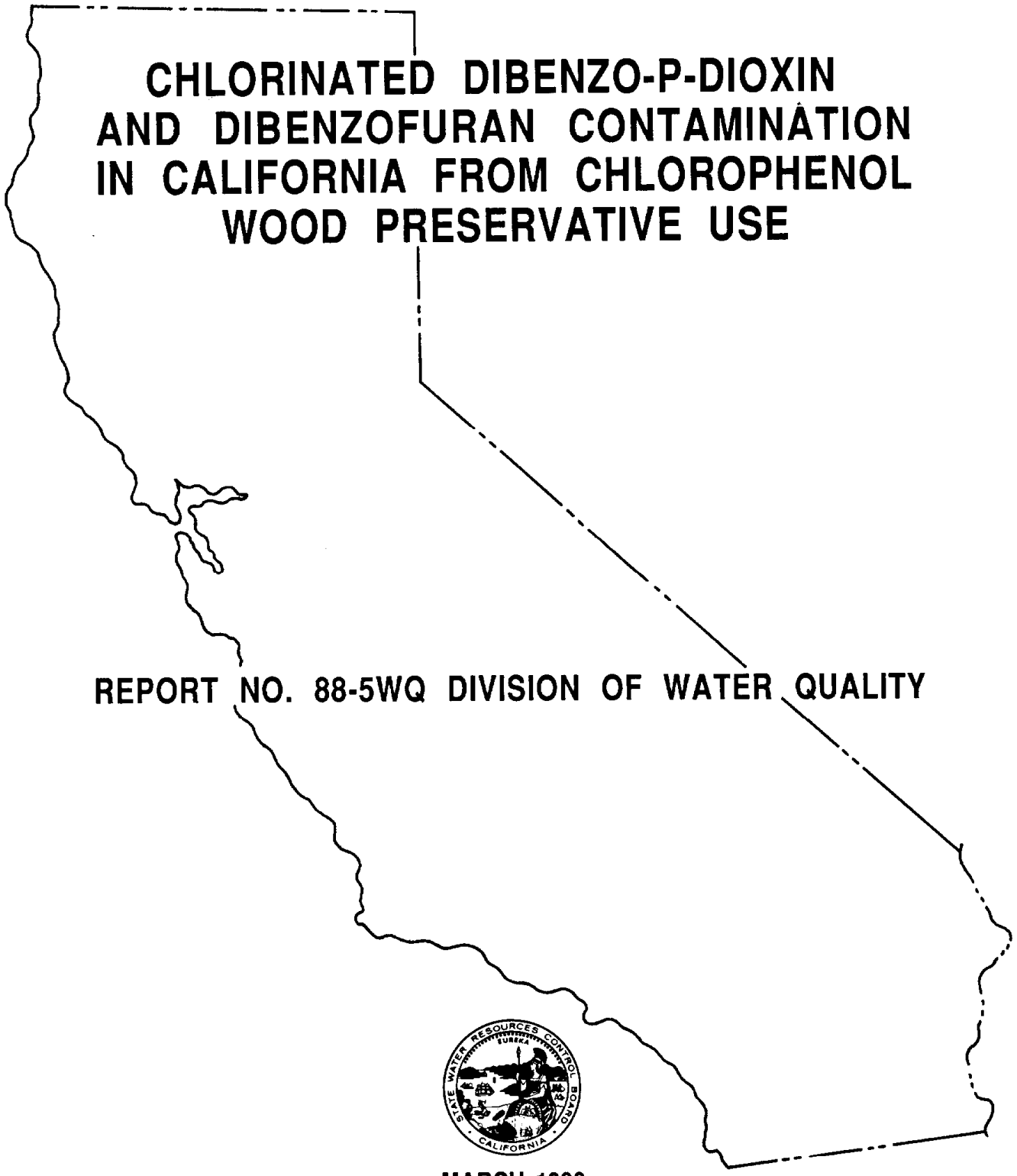

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REPORT NO. 88-5WQ DIVISION OF WATER QUALITY

Francis H. Palmer
Richard A. Sapudar
Judith A. Heath
Nancy J. Richard
Gerald W. Bowes

MARCH 1988
STATE WATER RESOURCES CONTROL BOARD

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PREFACE

This report is one in a series of reports issued by the State Water Resources Control Board on industrial and agricultural chemicals. These reports deal with priority chemicals of concern to water quality and the protection of beneficial uses of water in California. In February 1982, the State Board initiated an Industrial Chemicals program based on the premise that the production and use of chemicals should not occur at the expense of water quality protection.

Chemicals are of inestimable value to society, and most are considered relatively safe under normal conditions of use. There are some chemicals whose environmental and health effects have been proven harmful. The possibility that toxic chemicals in the environment can cause cancer in humans and severely impair the health of wildlife has led to increased action by government to foster the safe use and disposal of these chemicals.

The chronic effects of persistent chemicals (e.g., impaired growth and reproduction) may be more devastating in the long run than immediately apparent effects, such as fish kills. Preventative measures are invariably less costly to society than corrective actions required after toxic chemical pollution has occurred.

Some current chemical use and disposal practices may have an adverse impact on water quality. These activities can usually be modified to minimize adverse environmental effects. Where existing or potential water quality problems have been identified, the State Board will recommend appropriate measures to correct or prevent such adverse impacts.

LIST OF ABBREVIATIONS

Chemicals

CDD	Chlorinated dibenzo-p-dioxin
MonoCDD	Monochlorodibenzo-p-dioxin
DiCDD	Dichlorodibenzo-p-dioxin
TriCDD	Trichlorodibenzo-p-dioxin
TetraCDD	Tetrachlorodibenzo-p-dioxin
PentaCDD	Pentachlorodibenzo-p-dioxin
HexaCDD	Hexachlorodibenzo-p-dioxin
HeptaCDD	Heptachlorodibenzo-p-dioxin
OctaCDD	Octachlorodibenzo-p-dioxin
CDF	Chlorinated dibenzofuran
MonoCDF	Monochlorodibenzofuran
DiCDF	Dichlorodibenzofuran
TriCDF	Trichlorodibenzofuran
TetraCDF	Tetrachlorodibenzofuran
PentaCDF	Pentachlorodibenzofuran
HexaCDF	Hexachlorodibenzofuran
HeptaCDF	Heptachlorodibenzofuran
OctaCDF	Octachlorodibenzofuran
PCP	Pentachlorophenol
TetraCP	Tetrachlorophenol
TriCP	Trichlorophenol
NaPCP	Sodium pentachlorophenate
K-tetraCP	Potassium tetrachlorophenate
2,4-D	2,4-Dichlorophenoxyacetic acid
2,4,5-T	2,4,5-Trichlorophenoxyacetic acid
BAP	Benzo(a)pyrene
CCA	Chromated copper arsenate
DMBA	7,12-Dimethylbenz(a)anthracene
3MC	3-Methylcholanthrene
PCB(s)	Polychlorinated biphenyl(s)
PCDE	Polychlorinated diphenyl ether
TPA	12-0-Tetradecanoylphorbol-13-acetate

Terms

AAL	Applied Action Level
ADI	Acceptable daily intake
AHH	Aryl hydrocarbon hydroxylase
AWQC	Ambient water quality criteria
CERCLA	Comprehensive Environmental Response, Compensation, and Liability Act (Superfund)
CSF	Confidential statements of formula
CWA	Clean Water Act
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
GC	Gas chromatography
GC-MS	Gas chromatography-Mass Spectroscopy
HPLC	High performance liquid chromatography
LOEL	Lowest observed effect level
MFO	Mixed function oxidase
MS	Mass spectroscopy

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GLOSSARY

Most terms defined in the glossary are specific to usage in this report.

acute toxicity--involving a stimulus severe enough to rapidly induce an adverse response; in toxicity tests, a response observed in 96 hours or less is typically considered acute. Acute toxicity is most often reported in terms of lethality (e.g. LC50), but various other adverse effects may be measured (e.g. EC50).

adenoma--a benign neoplasm of glandular epithelial tissue.

adipose tissue--tissue in which fat is stored.

adsorb--the assimilation of gas, vapor, or dissolved matter onto a solid surface.

Ah receptor(aryl hydrocarbon receptor)--a soluble protein in the cell cytoplasm capable of binding an aromatic hydrocarbon molecule and inducing synthesis of gene products of the Ah locus.

Ah locus--gene complex responsible for the synthesis of aryl hydrocarbon hydroxylase (AHH) and several other enzymes.

aliphatic--a term applied to the "open chain" or fatty series of hydrocarbons; non-ring organic compounds.

alopecia--baldness;absence of hair from skin areas where it normally is present.

antigen--a substance capable of inducing the formation of antibodies in the blood.

Aroclor--trade name for a group of polychlorinated biphenyls; e.g. Aroclor 1242 indicates 12 carbon atoms and 42% chlorine by weight.

benign--not malignant; a benign tumor will not metastasize, a malignant tumor will.

bile--a fluid secreted by the liver that aids in digestion.

bilirubin--a reddish, yellow pigment in bile derived from hemoglobin during red blood cell (RBC) destruction.

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choline kinase--an enzyme that transfers a high energy group, such as phosphate, to choline, an amino alcohol and a member of the vitamin B complex.

chromatid--one of a pair of "sister" chromatids, identical connected nucleoprotein strands, products of replication of the parent chromosome, that are joined at the centromere and separate during cell division, each becoming a chromosome of one of the two daughter cells.

chronic toxicity--toxic effects from a prolonged exposure of an organism to sublethal amounts of a toxic substance, often one-tenth of the life span or more.

clean-up--laboratory purification of a sample before further analysis.

coelute--simultaneous desorption of two or more analytes from an analytical column such that they are not separated at the detector.

comedo(comedones-plural)--a plug in an excretory duct of the skin, containing microorganisms and keratin; also called a blackhead.

congener--refers to any one particular compound of the same chemical family; e.g. there are 75 congeners of chlorinated dibenzo-p-dioxin.

conjugate--a biochemical reaction product combining a foreign or natural compound or its metabolite with an endogenous carbohydrate, protein, or sulfur derivative.

cytochromes--any of a class of hemoproteins whose principal biological fuction is electron transport.

cytoplasm--the protoplasm (viscous, collodial semifluid) of a cell exclusive of the nucleus.

depurate--to be removed or reduced in concentration in a medium over time, as the result of a metabolic or physical process.

desorption--removal of a substance from an adsorbed state by physical or chemical process.

EC 50 (effective concentration)--the concentration of a substance in food and water at which 50% of the organisms treated exhibit the measured effect.

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gavage--feeding through a tube passed through the mouth into the stomach.

gene--the biological unit of heredity. A functional segment of DNA on a chromosome which codes and regulates production of one or more specific proteins.

genotoxicity--the effect of a substance that interacts with and alters DNA or RNA; when the DNA or RNA is replicated the alteration is carried on.

glucuronide--any compound containing glucuronic acid which is a tetrahydroxy-aldehyde acid.

glutathion-s-transferase--a family of enzymes that catalyzes glutathion conjugation.

gravid--pregnant;containing developing young.

half-life--the time in which the concentration of a substance will be reduced to one-half of its initial value through degradation or elimination from the medium.

hematologic--pertaining to blood.

hemoprotein--a conjugated protein containing heme as the prosthetic group, e.g. hemoglobin.

hepatic--pertaining to the liver.

humoral immunity--aquired immunity in which the role of circulating antibodies (immunoglobulins) is predominant.

hyperpigmentation--abnormally increased pigmentation.

hyperplasia--an abnormal increase in the size of a tissue or organ due to an increase in the number of normal cells.

ID_{50}--(immunological dose) a dose producing 50 percent suppression of the immune system.

immunoglobulin--an antibody synthesized by special lymphocytes (plasma cells) in response to the introduction of an antigen.

immunosuppression--the artificial inhibition of the immune system.

immunotoxic--quality of a substance which interferes with the immune system.

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liquid column chromatography--an analytical procedure where a sample or sample extract is passed through a column containing an absorbent which selectively entrains the analyte(s) of interest for later analysis.

log K_{ow}--logarithm of the partition coefficient between octanol and water for a given substance.

lymphoma--a general term applied to any neoplastic disorder of the lymphoid tissue.

mRNA(messenger ribonucleic acid)--a form of RNA in living cells that is responsible for carrying the genetic code transcribed from DNA to specialized sites within the cells for the synthesis of polypeptides.

malignant--an abnormal growth that tends to spread to other sites.

mass:charge ratio--in mass spectroscopy, the ratio of the mass of a fragment ion to its electronic charge.

mass spectrometer--an analytical instrument in which an analyte molecule is fragmented to produce a pattern of ions which is used for either identification or quantification.

maxilla--the iregularly shaped bone, composed of two maxillae joined together to form the upper jaw.

metabolite--any chemical substance produced by metabolism or by a metabolic process, e.g. breakdown products from biochemical reactions.

mexacarbamate--a carbamate pesticide.

micro-(u)--indicates one-millionth (10^{-6}); for example, there are one million (10^{6}) micrograms in a gram, or one billion (10^{9}) micrograms in a kilogram.

microbial degradation--the breakdown of compounds by microscopic organisms.

milli-(m)--indicates one-thousandth (10^{-3}); for example, there are one thousand (10^{3}) milligrams in a gram or one million (10^{6}) milligrams in a kilogram.

mixed function oxidases(MFO)--enzyme systems found predominantly in the endoplasmic reticulum of the liver which have a form of cytochrome P-450 as the terminal oxidase.

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peri position--chlorines in the 1,4,6,& 9 position on a CDD or CDF molecule.

photodegradation--chemical decomposition induced by light.

photolysis--chemical reaction involving bond-cleavage produced by exposure to light or ultraviolet radiation (adjective: photolytic).

photosensitize--to make an organism or chemical sensitive to light.

pico-(p)--indicates one-trillionth (10^{-12}); for example, there are one million million (10^{12}) picograms in a gram, or one quadrillion (10^{15}) picograms in a kilogram.

pleiotrophic gene--a gene that affects a number of different characteristics in a given individual.

porphyria--any of a group of disturbances of porphyrin metabolism characterized by marked increase in the formation and excretion of porphyrin precursors.

porphyrin--any one of a group of iron-free or magnesium-free cyclic tetrapyrrole derivatives which occur universally in the protoplasm.

predioxins--chlorinated 2-phenoxyphenols.

promoter--a substance which is not directly carcinogenic, but enhances effect of carcinogenic agents.

Salmonella typhimurium--a bacterial species used in mutagenicity tests.

sarcoma--a malignant neoplasm derived from connective tissue.

serum--blood plasma minus its clotting proteins.

sister chromatid exchange(SCE)--an exchange at one locus between the sister chromatids which does not result in an alteration of overall chromosome morphology.

sludge--the semiliquid precipitate of waste treatment processes, or settled residue in tanks or ponds used for chemical treatment, e.g. material found in lumber dip tanks at wood treatment facilities.

NOAEL	No observed adverse effect level
NOEL	No observed effect level
NSPS	New source performance standards
PSES	Pretreatment standards for existing sources
RCRA	Resource Conservation and Recovery Act
SNARL	Suggested no adverse response level
STLC	Soluble threshold limit concentration
TEF	Toxic equivalency factor
TTLC	Total threshold limit concentration

Units

ppm	parts per million
ppb	parts per billion
ppt	parts per trillion
ppq	parts per quadrillion
g/l	gram per liter
mg/l	milligram (10^{-3} grams) per liter (equal to ppm)
ug/l	microgram (10^{-6} grams) per liter (equal to ppb)
ng/l	nanogram (10^{-9} grams) per liter (equal to ppt)
pg/ml	picogram (10^{-12} grams) per milliliter (equal to ppt)
ug/m^3	microgram (10^{-6} grams) per cubic meter
pg/m^3	picogram (10^{-12} grams) per cubic meter
g/kg	gram per kilogram (equal to parts per thousand)
mg/kg	milligram per kilogram (equal to ppm)
ug/kg	microgram per kilogram (equal to ppb)
pg/g	picogram (10^{-12} grams) per gram (equal to ppt)

Government Agencies, Groups, and Private Industries

CAC	California Administrative Code
CARB	Calif. Air Resources Board
CAG	Carcinogen Assessment Group (U.S. EPA)
CDC	Center for Disease Control
CDWG	Chlorinated Dioxin Work Group (U.S. EPA)
CDHS	Calif. Department of Health Services
CVRWQCB	Central Valley Regional Water Quality Control Board
DWR	Calif. Department of Water Resources
U.S. EPA	United States Environmental Protection Agency
FDA	United States Food and Drug Administration
IARC	International Agency for Research of Cancer
NAS	National Academy of Science
NCI	National Cancer Institute
NIOSH	National Institute for Occupation Safety and Health
NRCC	National Research Council of Canada
NTP	National Toxicology Program
SCE	Southern California Edison
USFWS	United States Fish and Wildlife Service
WHO	World Health Organization

Symbols

>	greater than
≥	greater than or equal to
<	less than
≤	less than or equal to

ultraviolet (UV) spectroscopy--an analytical method that utilizes the fact that the amount of ultraviolet light absorbed by an analyte is a function of its concentration in solution.

uptake--absorption and incorporation of a substance by living tissue.

vehicle--the substance in which a compound is dissolved or mixed prior to dosing an animal with that compound.

wasting--gradual loss, decay, or diminution of bulk.

bioaccumulation--uptake, concentration, and retention of substances by an organism from its surrounding medium and from food.

bioassay--a test used to evaluate the relative potency of a substance by comparing its effect on a living organism with the effect of a standard preparation on the same type of organism.

bioavailability--the degree to which a drug or other substance is available to the target tissue after administration or to organisms in the environment.

bioconcentration--uptake and concentration of a substance from the surrounding medium through gill membranes or epithelial tissue.

bioconcentraion factor(BCF)--in standard tests, the ratio at equilibrium of the concentraion of a substance in the tissue of a test organism to its concentration in the surrounding medium. BCF is substance- and species-specific.

biotransformation--the series of chemical alterations of a compound which occur within an organism by enzymatic activity (sometimes causing the resulting compound to be more toxic, sometimes less.)

capillary column--a long open tube of small diameter having the inside wall coated with a thin film of stationary phase; used in gas chromatography for the separation of closely spaced peaks.

carcinogen--a cancer-producing substance.

carcinoma--a malignant growth derived from epithelial tissue and tending to infiltrate the surrounding tissue.

catabolism--any destructive process by which complex compounds are broken down into more simple substances.

caudal fins--tail fins of fish and aquatic mammals.

cell-mediated immunity--specific acquired immunity in which the role of small lymphocytes of thymic origin is predominant; it is responsible for resistance to infectious diseases caused by certain bacteria and viruses.

chloracne--a skin lesion resembling acne caused by exposure to chlorinated compounds.

EXECUTIVE SUMMARY

1. INTRODUCTION

In 1984, the State Board began a priority chemical investigation of certain chemicals used for wood preservation at California sawmills and wood treatment plants. Pentachlorophenol, one of the most widely used wood preservative fungicides, was given special attention, as it is known to contain highly toxic byproducts produced during its chemical manufacture. These contaminants include chlorinated dibenzodioxins (CDDs) and a related group of chemicals, chlorinated dibenzofurans (CDFs). Chemical identification of these substances is extremely difficult, in part because there are so many of them (75 different CDDs and 135 possible CDFs). Only 15 of these 210 compounds (6 CDDs and 9 CDFs) are considered highly toxic. The most toxic compound is commonly referred to as "dioxin" or 2,3,7,8-tetrachlorodibenzodioxin. As "dioxin" has been studied most extensively, much of what has been estimated about the other CDDs and CDFs is based on knowledge of this compound.

The CDDs and CDFs have never been intentionally manufactured. They are only produced as reference standards which are required for chemical analysis. In addition, CDDs and CDFs are known to occur as byproducts of chemical synthesis, from electrical equipment fires, and from municipal solid waste incinerators. The CDDs and CDFs have received widespread media attention because of several incidents involving human exposures. These events include the use of the herbicide Agent Orange in Vietnam, a chemical plant explosion at Seveso, Italy, CDD-contaminated oil used for dust control in Missouri, and CDF-contaminated rice oil poisoning incidents in Japan and Taiwan.

The State Board study described in this report was designed to determine which, if any, of the 15 most toxic CDDs and CDFs were present at sawmills and wood treatment plants in California. In order to perform the difficult chemical analysis, split samples were sent to three laboratories in the United States and Sweden. Several of the 15 most toxic CDDs and CDFs were detected in samples of soil, sawmill sludges and liquids, commercial pentachlorophenol formulations, and crystals formed during wood pressure treatment.

edema--the presence of abnormally large amounts of fluid in the intercellular tissue spaces of the body.

embryotoxicity--stillbirth or in utero death during the embryonic stage before the placenta is completely formed, which in humans is approximately the first 8 weeks after conception.

endoplasmic reticulum--an ultra microscopic organelle of nearly all cells of higher plants and animals consisting of a more or less continuous system of membrane-bound cavities that ramify throughout the cytoplasm of the cell.

enzyme induction--increased activity of the enzyme systems upon exposure to chemicals.

epidemiological--relation of the various factors determining the frequency and distribution of diseases within a given population.

epoxide--cyclic ethers; an atom of oxygen bound to two separate carbons which are linked, forming a three-membered ring.

eutrophication--the natural process of aging of bodies of water resulting in an increase in mineral and organic nutrients such as nitrogen and phosphorus, and reduced levels of dissolved oxygen. Eutrophic lakes may be characterized by algal or bacterial blooms and diminished fish life.

extraction--separation and isolation of analytes from a sample matrix, usually through the use of solvents.

femto-(f)--indicates one-quadrillionth (10^{-15}); for example, there are one million billion (10^{15}) femtograms in a gram, or one billion billion (10^{18}) femtograms in a kilogram.

fetotoxicity--stillbirth or in-utero death during the fetal (post-embryonic) stage.

fibrosarcoma-a malignant neoplasm derived from fibrous connective tissue.

fractionation--a step in sample preparation for analysis which separates the analytes contained in a sample into multiple fractions which have similar physical and chemical properties.

frameshift mutation--addition or deletion of base pairs on the DNA molecule. If the number of base pair changes is not a multiple of three, the amino acid sequence of the proteins coded after the mutation is drastically changed.

3. AQUATIC TOXICOLOGY

In addition to toxic effects occurring at very low (parts per trillion) concentrations, the most striking aspect about the effect of "dioxin" on aquatic life is that toxic reactions are not observed until 5 to over 100 days after exposure. An amount as low as 5.6 parts per trillion has been shown to be lethal to salmon with other toxic effects observed as low as 0.1 parts per trillion. The CDDs and CDFs also are bioconcentrated to a high degree in aquatic organisms. The highest reported bioconcentration factor is approximately 9,000 for both rainbow trout and mosquito larvae. The most toxic CDDs and CDFs are also most preferentially bioconcentrated.

As this report went to press, the State Board learned of new toxicity and bioconcentration information obtained from a recent chronic study. Published in January 1988, the study examined the effects over a 56-day period of very low levels of the most toxic CDD and most toxic CDF on rainbow trout. Levels as low as 38 parts per quadrillion of the CDD had significant adverse effects on survival and growth. CDF levels as low as 0.9 parts per trillion reduced growth and 4 parts per trillion reduced survival. Bioconcentration factors by rainbow trout also were higher than previously reported: 39,000 for the CDD and 6,000 for the CDF.

4. MAMMALIAN TOXICOLOGY

Both CDDs and CDFs are absorbed and concentrated by humans and laboratory animals. The half-life of the most toxic CDD was over five years in a human volunteer, in contrast to shorter half-lives (10 to 40 days) in laboratory animals.

The most toxic CDD is also extremely variable in lethality, depending on animal species. For example, it takes approximately 5,000 times as strong a dose to kill a hamster as a guinea pig. As with aquatic animals, death in mammals is delayed after a single lethal dose, typically between 5 and 45 days. Death occurs after a period of wasting away.

In addition to lethality, these compounds also produce long term effects. Studies with laboratory animals have shown that the most toxic CDD causes reproductive (teratogenic) and fetal (fetotoxic) defects at very low exposure levels. These effects have not, however, been observed to date after accidental human exposure. Studies of the most toxic CDD and of a mixture of two other toxic CDDs have shown these compounds to be strong animal carcinogens. The U.S. Environmental Protection Agency (EPA) has rated the most

in utero--within the uterus.

in vitro--within an artificial environment, e.g. biochemical studies in laboratory glassware.

in vivo--within a living body.

initiation--interaction of a carcinogen with a normal cell to produce a cancerous or precancerous cell.

integumentary system--an enveloping layer (as a skin or membrane) of an organism or one of it parts.

intraperitoneal--injection into the abdominal cavity that is lined by a serous sac (peritoneum).

isomer group--a group of structurally related chemicals (isomers) with the same molecular formula, e.g. there are eight isomer groups of CDDs, monochlorinated through octachlorinated.

isomers--chemical compounds that have the same molecular formula but different molecular structures or different arrangements of atoms in space; refers to substances which belong to the same homologous class; e.g. 22 isomers constitute the homologue of tetraCDD.

kinetic--refers to the processes and rates of chemical reactions.

LC_{50}--the lethal concentration (LC) of a toxicant in food or water to 50% of the exposed population.

LD_{50}--the lethal dose (LD) of a toxicant to 50% of the exposed population.

leachate--water that has percolated through soil containing soluble substances and that contains certain amounts of these substances in solution.

lignin--a polysaccharide which, in connection with cellulose, forms the cell wall of plants and wood.

lipid--fatty material characterized by the presence of fatty acids or their derivatives and by their solubility in non-polar solvents.

lipophilic--readily soluble in lipid.

This report provides three examples of contamination by pentachlorophenol in California: at Visalia, Selma, and Oroville. At the Visalia site, a plume of organic solvents transported pentachlorophenol, CDDs and CDFs into both the shallow and deep aquifers. Contamination of the deeper aquifer was especially worrisome since the City of Visalia's drinking water wells were located downstream of the site. High levels of CDDs and CDFs were detected in soil samples at Selma while extensive pentachlorophenol contamination of ground water has occurred near Oroville.

7. CALIFORNIA WATER RESOURCES CONTROL BOARD STUDY

Based on a report of high levels of CDFs found in dip tanks at two Swedish sawmills, the State Board investigated wood treatment facilities in California to determine if CDDs and CDFs were also present.

When CDDs and CDFs are found, they usually occur as a complex mixture of different compounds. The degree of difficulty of chemical analysis for CDDs and CDFs depends on the type of analysis performed. The easiest method is group analysis. For example, one group of CDDs contains six chlorine atoms. There are actually ten different CDDs with six chlorines, but, by measuring only the group, the analysis is simplified.

Testing for individual CDD and CDF compounds is much more difficult. For example, of the ten different compounds in the six chlorine group of CDDs, three are highly toxic. The most accurate approach to evaluate their toxicity would be to measure the individual concentrations of these three highly toxic compounds.

The State Board study tested both the simpler group approach as well as individual compound analysis. In the group analysis phase, 13 samples -- soil (4), sludge (4), dip tank liquid (2), and commercial pentachlorophenol (3) -- were examined for presence of CDDs and CDFs. Significant concentrations of these groups of compounds were detected in all samples, with the highest concentrations found in the commercial formulations and dip tank sludges.

In the subsequent individual compound analysis phase, 12 samples from four sites (three sawmills, and one wood pressure treatment plant) were analyzed for the 15 most toxic CDDs and CDFs. Typically, at least three of the six toxic CDDs and seven of the nine CDFs were present in sawmill sludges and commercial mixtures. A noteworthy sample was obtained at a pressure treatment plant, where the

mutagen--a physical or chemical agent that alters DNA or RNA.

mutagenicity--the ability of a substance to alter DNA or RNA.

nano-(n)--indicates one-billionth (10^{-9}); for example, there are one billion (10^{9}) nanograms in a gram, or one trillion (10^{12}) nanograms in a kilogram.

necrosis--death of tissue.

neoplasm--new and abnormal growth, such as a tumor, that may be either benign or malignant.

neoplastic nodule--abnormal swelling or protuberance.

neuropathy--a general term denoting functional disturbances and/or pathological changes in the peripheral nervous system.

NOAEL--no observed adverse effect level; synonymous with NOEL.

NOEL--no observed effect level; the highest measured continuous concentration of an effluent of a toxicant that does not cause health effects or clinical signs on a test organism.

nuclear magnetic resonance--an analytical method for identification of atomic constituentsof chemicals, using knowledge of absorption of electromagnetic radiation at a precise frequency by the atomic nucleus.

oligotrophic--a body of water with a poor supply of nutrients and a low rate of formation of organic matter by photosynthesis.

opercular--pertaining to an operculum which is a lid or flap of skin covering an opening or orifice, e.g. the gill cover of fishes.

organic--denoting chemical substances containing the element carbon.

pancytopenia--deficiency of all cell elements of the blood; aplastic anemia.

parenchymal cells--cells in loose connective tissue whose function is to pack the space between organs.

partition coefficient--the ratio of a chemical distributed between two parts of a system such as octanol and water or sediment and water.

RECOMMENDATIONS

1. Sawmill sludges and soils should be analyzed for the presence of CDDs and CDFs prior to disposal.

   The CDDs and CDFs previously concentrated in dip tank sludges will remain until the tanks are cleaned. Before disposal, these sludges should be analyzed for potential presence of CDDs and CDFs. If these compounds are present, sludge disposal by land or low temperature burning should be avoided. These materials should be held in interim storage until an effective means of destruction is identified and is available.

2. Wood treatment plants should improve management practices to isolate crystals of pentachlorophenol formed after treatment.

   Crystals (or "bloom") formed on lumber after pentachlorophenol pressurized treatment contain high levels of toxic CDDs and CDFs. During sampling by State Board staff, it was observed that some of this material falls to the ground during normal operating procedures. Plant operations should be improved to prevent environmental contamination by these crystals.

3. The highest priority should be given to isolating chlorinated dibenzodioxins and dibenzofurans from the environment and destroying them.

   Over 100 million dollars has been expended worldwide for research on the most toxic CDD. Nevertheless, many questions regarding toxicity and environmental fate of CDDs and CDFs still exist. Effective means to safely degrade these compounds, such as high temperature incineration or other methods, must be developed as rapidly as possible.

4. Interim on-site storage of CDD and CDF-containing materials is recommended until effective means of destruction are developed.

   Mobility and availability of CDDs and CDFs are dependent upon site specific soil types and characteristics, annual rainfall, plant and animal populations, and bioavailability. CDDs and CDFs should therefore not be placed in landfills. If, in the future, on-site land treatment is proposed, methods must be specifically designed for each site to avoid human or environmental exposure.

soil column--a vertical column of soil usually a core sample, which displays horizontal layers of soil material.

solubility--the amount of a substance that can be dissolved in a given amount of solvent, normally expressed as mg/l.

soxhlet extraction--a repetitive extraction and distillation procedure for extracting analytes from a solid sample matrix.

static--at rest or in equilibrium; not dynamic.

steady state--a stable equilibrium condition of a system in which change in one direction is continually balanced by change in another.

structure-activity relationship--the biochemical activity of a compound related to its structure.

subchronic toxicity--toxic effects produced by a test compound during an exposure of intermediate duration usually lasting about three months.

subclinical--without clinical manifestations; said of the early stages, or a slight degree, of a disease.

subcutaneous--beneath the skin.

substrate--a substance upon which an enzyme or catalyst acts.

T-lymphocyte--a thymus dependent lymphocyte (a mononuclear white blood cell).

technical grade chemical--a chemical that has not been purified after production and may contain many impurities.

teratogenic--producing non-lethal morphological or functional changes in the fetus.

thymic atrophy--wasting away or decrease in size of the thymus.

thymus--a bilobed organ, located in the lower neck, that plays a role in the immune mechanism of the body.

triglyceride--a compound consisting of three molecules of fatty acid esterified to a glycerol.

turbinate--shaped like a cone; a turbinate bone located in the nasal passages.

9. Interim advisories for highly toxic CDDs.

Advisory limits have been proposed by the U. S. Government, by other states, and by the province of Ontario, Canada, for drinking water, fish flesh, and soil cleanup. Although not the focus of this State Board report, the starred (*) levels listed below can serve as interim guidelines for California until advisories are established by the California Department of Health Services. It should be noted that some of these advisories are at or below the current practical detection limits for these compounds.

a. 2,3,7,8-tetraCDD (the most toxic CDD)

i. Drinking Water (protection of human health)

U.S. EPA (1984)	2.2×10^{-4} ppt*	(0.2 parts per quadrillion)
National Academy of Sciences (1977)	0.7 ppt	
New York State (Ground Water-drinking water supply) (1987)	3.5×10^{-2} ppt	(35 parts per quadrillion)

ii. Fish Flesh

U. S. FDA (1983)	50 ppt*
Province of Ontario (1986)	20 ppt
Michigan (1986)	10 ppt
New York (1987)	10 ppt

iii. Soil Cleanup Level

United States Centers for Disease Control (Atlanta, GA) (1984)	1 ppb	(site-specific for Times Beach, Missouri)

b. hexaCDD (six-chlorine CDD) - Drinking Water

U.S. EPA (1985)	5.5×10^{-3}* ppt	(5.5 parts per quadrillion)

TECHNICAL SUMMARY

1. INTRODUCTION

Highly toxic compounds were found in products and environmental samples at selected California sawmills and wood treatment plants. These were chlorinated dibenzodioxins and chlorinated dibenzofurans ("CDDs" and "CDFs"). These classes of compounds include 2,3,7,8-tetrachlorodibenzodioxin, which is popularly referred to as "dioxin".

Structures of these compounds are shown in Figure 1. Dioxin has received widespread press coverage because it was a contaminant in Agent Orange, an herbicide used in Vietnam. It was detected in the streets of Times Beach, Missouri, and traced to contaminated oil used for dust control. The town was evacuated and bought out by the U. S. Government after the Centers for Disease Control determined that the 2,3,7,8-tetraCDD concentrations in soil represented an unreasonable risk to humans. Chlorinated dibenzofurans (CDFs) are contaminants in polychlorinated biphenyl (PCB) formulations. Both CDFs and PCBs contributed to significant human health problems in Japan and Taiwan. Rice oil had been accidentally contaminated with high concentrations of both compounds and was consumed by humans.

As shown in Figure 1, the chlorinated dibenzodioxin and dibenzofuran molecules each can contain from one to eight chlorine atoms. Since these can be arranged in a variety of ways, up to 75 CDDs and 135 CDFs are possible (Table 1). A mixture having both CDDs and CDFs theoretically could contain 210 individual compounds. The CDDs and CDFs having four, five, six, or seven chlorine atoms, four of which are in the 2,3,7, and 8 positions, are considered to be significantly toxic to mammals. The number of these is fifteen: six CDDs and nine CDFs (Table 2). The two eight-chlorine containing ("octa-") CDDs and CDFs also have four 2,3,7,8-substituted chlorine atoms. However, the octaCDDs and CDFs are believed to have low toxicity and in this report are not considered in the hazard evaluations of samples containing them.

CDDs and CDFs are not produced intentionally, except as reference standards for chemical analysis. They appear, for example, as by-products of chemical synthesis, electrical equipment fires, and municipal incineration of solid wastes. They are contaminants of chlorophenol wood preservatives. In California, approximately 100 sawmills and wood treatment plants have been in operation or exist today. Almost half of these have used chlorophenol wood preservatives. These chemicals and their contaminants are present at an undefined number of sites, regardless of whether or not the plants are still operating.

2. ENVIRONMENTAL FATE

As a group, the CDDs and CDFs share three characteristics that make them long-lived in the environment: very low water solubility, high affinity for soil and sediment and resistance to breakdown. However, as individual compounds, the CDDs and CDFs exhibit wide diversity. For example, the eight chlorine CDD is about 100,000 times less soluble than the CDDs containing four chlorine atoms. The combination of very high toxicity and very low water solubility has made the measurement and modeling of CDDs and CDFs in the environment a difficult task. However, recent work has shed some light on a number of processes that may affect the persistence of these compounds in the environment. These include the following:

a. On soil surfaces, CDDs and CDFs can be both formed and broken down by sunlight. For example, they can be formed from the joining of two pentachlorophenol molecules, while more highly chlorinated compounds can be converted to lower chlorinated ones. Under certain conditions, the lower chlorinated CDDs and CDFs that are formed from such breakdown conversions can be more toxic than more highly chlorinated parent compounds.

b. Naturally occurring micro-organisms will not significantly breakdown CDDs and CDFs.

c. Despite having low vapor pressures, CDDs and CDFs can be transported from water and soil to the air. Detection of these compounds at clean sites is therefore strongly suggestive of atmospheric deposition.

d. CDDs and CDFs can migrate to ground water if organic solvents are also present. In the absence of organic solvents, they are not expected to migrate significantly unless "channels" such as cracks in rocks are present.

e. CDDs and CDFs will bind strongly to suspended matter in water. The major "sinks" for these compounds in water are sediments, particulates, and living organisms.

f. Because of the extremely low water solubility of CDDs and CDFs, water-based leachate tests designed to simulate conditions in a municipal landfill are not likely to detect their presence.

TABLE 1

NUMBER OF COMPOUNDS IN CHLORINATED DIBENZODIOXIN AND DIBENZOFURAN ISOMER GROUPS

Isomer Group	Number of Compounds in Isomer Group
CDDs	
1. Monochlorodibenzodioxin (monoCDD)	2
2. Dichlorodibenzodioxin (diCDD)	10
3. Trichlorodibenzodioxin (triCDD)	14
4. Tetrachlorodibenzodioxin (tetraCDD)	22
5. Pentachlorodibenzodioxin (pentaCDD)	14
6. Hexachlorodibenzodioxin (hexaCDD)	10
7. Heptachlorodibenzodioxin (heptaCDD)	2
8. Octachlorodibenzodioxin (octaCDD)	1
TOTAL CDD COMPOUNDS	75
CDFs	
1. Monochlorodibenzofuran (monoCDF)	4
2. Dichlorodibenzofuran (diCDF)	16
3. Trichlorodibenzofuran (triCDF)	28
4. Tetrachlorodibenzofuran (tetraCDF)	38
5. Pentachlorodibenzofuran (pentaCDF)	28
6. Hexachlorodibenzofuran (hexaCDF)	16
7. Heptachlorodibenzofuran (heptaCDF)	4
8. Octachlorodibenzofuran (octaCDF)	1
TOTAL CDF COMPOUNDS	135
CDD AND CDF TOTAL	210

toxic CDD as the most potent animal carcinogen ever tested. However, there is little conclusive evidence from human exposure to date that this compound is linked to human cancer. A recent newspaper account in the New York Times (December 9, 1987) noted that EPA may reduce the estimate of CDD potency by a factor of 16. If this EPA rating system estimate does change, CDD will still be the most toxic carcinogen known. At the new estimate, the "safe" daily dose would be raised to 0.1 parts per quadrillion per day based on body weight (A part per quadrillion is one divided by 10^{15}).

5. CRITERIA AND STANDARDS

Criteria and standards have been developed primarily for the most toxic CDD. For example, the U. S. Food and Drug Administration in 1983 set a safe level of 25 parts per trillion in fish for human consumption as long as fish was not consumed more than twice a month. The U. S. Centers for Disease Control recommended a site specific cleanup level of 1 part per billion in soil. There is considerable debate in the scientific community over whether the 1 part per billion level for soil cleanup is too conservative (too safe) or not safe enough.

The EPA currently considers the most toxic CDD such a strong carcinogen that the one in one million risk level is set below the current chemical detection limit. This water criterion of 0.013 parts per quadrillion is based on a daily intake by a 70 kilogram man of 2 liters of water and 6.5 grams of fish or shellfish.

6. WOOD TREATMENT PRACTICES AND CALIFORNIA SITE CONTAMINATION

Pentachlorophenol and similar compounds have been used routinely for decades at sawmills and wood treatment facilities in California. Wood is typically treated by either dipping it in tanks containing the preservative solution, by spraying, or by forcing the solution under pressure into the wood. The latter method is used at wood treatment plants to provide long lasting protection. In contrast, sawmills use the dipping or spraying methods as a shorter term means to protect the surface from fungal growths that stain the wood and degrade its market value. Typically, the areas where wood is treated have been contaminated by the treatment chemical. Where pentachlorophenol has been used, the contaminants have included CDDs and CDFs. Because of their environmental persistence, these compounds may be present many years after the use of pentachlorophenol has ceased.

2,3,7,8-TetraCDD is the most potent animal carcinogen ever evaluated in the laboratory. EPA has estimated that this compound is approximately 20 and 50 times more potent than the next two highest-ranked carcinogens (a mixture of two hexaCDDs and Aflatoxin B_1, respectively). It is 50 million times more potent than trichloroethylene (TCE) or vinyl chloride. This 2,3,7,8 four chlorine-containing compound also is highly acutely toxic to certain animal species. A single feeding of one part to one billion parts body weight will kill half of a guinea pig test population.

The findings of dramatic CDD acute toxicity and carcinogenicity in animals contrasts with the lack of comparable findings in humans. Over one hundred million dollars has been spent over the last few decades studying the toxicity and fate of principally one compound, 2,3,7,8-tetraCDD. Large gaps in knowledge still exist. The most prudent approach at this time should be minimizing CDD and CDF entry into the environment. This is an alternative to continuing to spend large sums of money on research that produces as many questions as answers.

The present State Board study detected CDDs and CDFs at sawmills and wood treatment plants in soils and dip tank liquids and sludges. CDDs and CDFs were present where pentachlorophenol had been used for wood preservation. Most of the toxic CDD and CDF compounds listed in Table 2 were detected in all samples. To our knowledge, this is the first study in the United States which has identified the fate of individual 2,3,7,8-substituted CDDs and CDFs in chlorophenol wood preservatives after their use. The analytical chemistry necessary to perform such detailed trace analysis involved three laboratories in the United States and Sweden.

2. ENVIRONMENTAL FATE

The anticipated stability and distribution of CDDs and CDFs depends upon the individual compound, environmental conditions, and the nature of experiments designed to predict its environmental fate. Available data show that CDDs and CDFs can be (1) formed in the environment; (2) degraded; (3) remain unchanged; and (4) migrate through soil to ground water. The most useful predictive information comes from actual field measurements as well as laboratory experiments which have been constructed to simulate field conditions closely. The fairly sizable number of environmental fate experiments, especially in the area of light-related effects is confusing, but a general understanding of this fate is beginning to emerge.

crystals or "bloom" formed on the surface of treated lumber contained five of the most toxic CDDs and eight of the most toxic CDFs. This information indicates that highly toxic CDDs and CDFs can be present, often at significant concentrations, as contaminants at sawmills and wood treatment plants.

8. HAZARD EVALUATION

The approach used in this report is based on an interim method, published in 1986 by the EPA, to evaluate the toxicity of CDD and CDF mixtures. It follows the premise that these different compounds follow similar toxicological pathways and that their toxic effects in mixtures are additive.

Each of the 15 highly toxic CDDs and CDFs has a different estimated toxicity. The EPA approach is to assign the most highly toxic CDD (the "dioxin") a toxicity value of 1.0 units, while the remaining 14 are given values ranging from 0.001 to 0.5 units, based on available toxicity information.

The "total" toxicity of a particular mixture of CDDs and CDFs is then calculated by multiplying the toxicity value of each separate CDD or CDF by its concentration in the sample. This step is performed for each of the highly toxic CDDs and CDFs and the results are added to obtain a total toxicity concentration for the mixture. Using this method, the highest relative toxicity concentration determined in a commercial pentachlorophenol formulation was 290 parts per billion. In sawmill dip tank sludge, the relative toxicity concentration ranged from 27 to 330 parts per billion. In the crystals formed after pressure treatment, the relative toxicity concentration was calculated to be 100 ppb.

Characterization of the "total" toxicity of CDDs and CDFs in mixtures by this method allows for estimation of site specific potential hazards as well as options for remedial action. The report recommends that remedial action assessment be based upon the "Decision Tree" approach developed by the California Department of Health Services. At some sites, moving the material may create more of a hazard than on-site storage of CDD and CDF containing materials isolated from humans and the environment. The latter approach may be the most effective interim measure until acceptable methods of CDD and CDF destruction become available.

Little information is available to predict the stability and extent of distribution of CDDs and CDFs once they have evaporated from water and land surfaces. They can exist in vapor and adsorb to particulate matter in air. The presence of CDDs and CDFs in lake sediments located on a Lake Superior island indicates that these compounds can be atmospherically transported and subsequently redeposited.

Persistence and Movement in Soils and Sediments

As noted above, CDDs and CDFs have been detected in ground water, probably by being transported through soil by organic solvents. CDDs have reached a depth of 30 meters in Florida, and 16 meters in California. In the absence of organic solvents, CDDs and CDFs are not expected to move downward to any great extent.

Migration of these compounds at waste disposal and land treatment sites cannot be predicted accurately by spiking solvent-free soil with CDDs and CDFs, and rinsing the soil with water. A standard soil leachate test specified by RCRA for dioxin-containing wastes requires use of water to leach CDDs from soil. A more accurate test would employ a mixture of water and organic solvents. The more accurate test would increase the amounts of CDD and CDF compounds extracted from soil and thereby their concentration in a leachate test. This in turn would increase the likelihood that CDD and CDF contaminated soil would not be acceptable under RCRA treatment standards. At present, the RCRA treatment standard requires that wastes found to contain any tetra-, penta-, or hexaCDDs or CDFs at concentrations of 1 ppb or more in a standard leachate test be treated before land disposal.

CDDs and CDFs are also expected to adsorb strongly to sediments and suspended particulate matter in water. As a result, and because of their stability, they are expected to be highly persistent in these associations. In aquatic systems, therefore, the major "sinks" for CDDs and CDFs will be sediments, suspended particulates, and biota.

Plant Uptake

Measurements of CDDs and CDFs have shown that these compounds are concentrated in aquatic plant extracts. This can be interpreted to show that CDDs and CDFs are taken up and concentrated by aquatic plants. However, an undetermined amount of this material may be adsorbed onto the plant surface rather than being absorbed by the plants. Bioaccumulation figures for these compounds in aquatic plants should be interpreted with some reservation, especially for unicellular phytoplankton where the surface area is large compared to the internal volume. The distinction is not important to zooplankton or fish consumers of aquatic plants:

5. The California Site Mitigation Decision Tree Manual (Decision Tree) should be used as guidance for clean-up of CDD and CDF-contaminated sites.

   The Decision Tree process, published by the California Department of Health Services, consists of five elements: (1) preliminary site appraisal; (2) site assessment; (3) risk appraisal; (4) environmental fate and risk determination; and (5) development of site mitigation strategies and selection of remedial action.

6. Estimates of the concentrations of the most highly toxic CDDs and CDFs in contaminated materials should be made by following procedures described in this report.

   Considering the complexity and expense of analyzing for 210 individual CDDs and CDFs, analysis should be focused on the eight groups. Then the "total" toxicity of the most toxic CDD and CDF compounds in soil and dip tank samples can be estimated by using the percentage of highly toxic compounds calculated in this report. This will greatly simplify analysis for CDDs and CDFs by identifying only the four, five, six, and seven chlorine groups for each of these two compound classes.

7. Estimation of the toxicity of CDD and CDF mixtures should follow the U.S. EPA "toxicity equivalency factor" approach.

   As an interim approach to estimating the toxicity of samples containing CDD and CDF complex mixtures, the U.S. EPA has recommended a system based on multiplying the concentrations of individual highly toxic CDDs and CDFs by respective potency factors. These factors are based on both carcinogenicity and other toxicity test values of various CDDs and CDFs relative to the most toxic CDD.

8. EPA should develop a national strategy for identifying chemicals (or classes of chemicals) that may cause toxicity beyond the normal 96 hour acute test period.

   For chemicals thus identified, EPA should recommend observation periods for acute aquatic toxicity be extended from the current 96 hour standard bioassay test to at least 30 days beyond the acute test period. These recommendations follow observations of toxic effects induced by CDDs and CDFs up to one month after the initial exposure, when mortality did not occur within the standard 96 hour test period.

Adverse toxic reactions most likely would have been observed at lower concentrations of 2,3,7,8-tetraCDD than reported, if the bioassays had been the continuous-flow type. Here, both water and toxic chemicals are renewed on a continuing basis. This simulates many natural situations. Effects may be seen at lower water concentrations because of the continuous renewal of water containing the toxicant.

Few CDD chronic studies have been reported. CDF toxicity has been estimated only in studies where CDF-contaminated food was provided to the fish.

CDDs and CDFs accumulate in aquatic organisms. The highest reported bioconcentration factor for 2,3,7,8-tetraCDD is approximately 9,000 for both rainbow trout and mosquito larvae. This is possibly an underestimate of bioconcentration potential due to the static test condition.

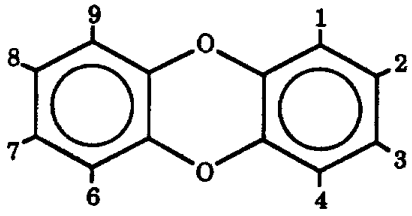
One investigator exposed fish to a mixture of CDDs and CDFs containing from four to eight chlorine atoms. With few exceptions, those compounds having chlorines at the 2,3,7, and 8 positions were selectively concentrated by the fish. Others have observed that compounds with chlorine atoms in other positions also were accumulated by fish. In these latter experiments, the 2,3,7, and 8 compounds were not present. The extent to which molecular configuration influences uptake needs clarification.

Studies of elimination of CDDs and CDFs from fish that have been exposed to these compounds in water showed: (1) rate of elimination decreases with increasing chlorination of the compound; and (2) for the same degree of chlorination, CDFs are depurated at a greater rate than CDDs.

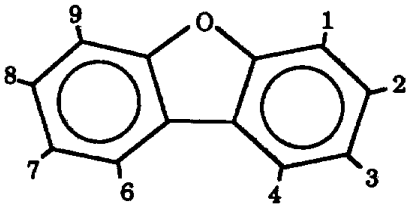
Subsequent to completion of this State Board report, data were published that showed higher toxicity and bioconcentration than previously reported. This new study, published in January 1988, described chronic effects of 2,3,7,8-tetraCDD and 2,3,7,8-tetraCDF on rainbow trout. The experiment was a 56-day flow-through test with 28 days of exposure followed by 28 days of depuration. At 38 parts per quadrillion 2,3,7,8-tetraCDD, the lowest concentration tested, significant adverse effects were observed on growth and survival. Because effects were determined at the lowest level, a no observed effect concentration (NOEC) for this CDD could not be derived. At 0.9 parts per trillion (ppt) 2,3,7,8-tetraCDF, reduced growth effects were reported and reduced survival was observed at 4 ppt. NOEC values were 0.4 ppt for growth and 1.8 ppt for survival for this CDF. While the

FIGURE 1

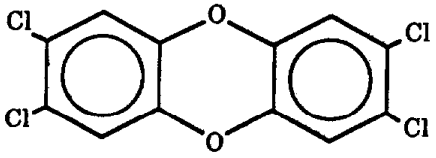
CHEMICAL STRUCTURES



Dibenzo-p-dioxin



Dibenzofuran



2,3,7,8-Tetrachlorodibenzo-p-dioxin
(2,3,7,8-TetraCDD)
("Dioxin")

NUMBERS ON STRUCTURES REFER TO LOCATION WHERE CHLORINE ATOMS CAN BE ATTACHED

CDDs and CDFs can be expected to be distributed in the body in proportion to the amount of fat content of a particular tissue. In both laboratory animals and humans, highest concentrations are found in adipose tissue and liver.

Laboratory experiments with 2,3,7,8-tetraCDD and a CDF mixture have shown that these chemicals can move through the placenta. One study also showed that CDFs are transferred to the offspring in greater amounts through milk, compared to transport through the placenta.

Laboratory studies have shown that animals can transform absorbed 2,3,7,8-tetraCDD. Unidentified transformation products have been detected principally in the bile and urine. Depending on the compound, metabolites can be either more or less toxic than the parent from which they are derived. EPA has noted that metabolism of 2,3,7,8-tetraCDD appears to be mostly a detoxification process which produces metabolites less toxic than the parent compound.

Acute, Subchronic, and Chronic Toxicity Effects

As noted, one of the most acutely toxic substances known is 2,3,7,8-tetraCDD. However, species sensitivity can differ significantly. The male hamster is approximately 8000 times less sensitive than the male guinea pig in a short-term lethal dose test. When 2,3,7,8-tetraCDD is fed to animals in acutely toxic doses, death is delayed and may take from 5 to 45 days. During this period, weight loss occurs with the animals exhibiting a characteristic "wasting away" appearance. This compound also induces liver damage in most species. The immune system is adversely affected in all species tested. Thymic atrophy is the principal change. The spleen, lymph nodes, and bone marrow may be affected. Susceptibility to bacterial infection is increased, and antibody production decreased.

One experiment focused on the relative effects of technical grade pentachlorophenol (PCP) and its contaminants on immunosuppresion. The contaminants included chlorinated diphenyl ethers, phenoxy phenols, dibenzodioxins and dibenzofurans. Technical grade PCP contained 86 percent pentachlorophenol. This produced a dose-related decrease in antibody response. In contrast, analytical grade PCP, which was greater than 99 percent pure, had no effect. Neither did the chlorinated phenoxy phenol or diphenyl ether components. The experimenters concluded that a significant amount of the immunosuppression was caused by the CDDs and CDFs.

Most human exposures to CDDs and CDFs have occurred either occupationally or accidentally, and concurrently with exposure with other chemicals. In these situations the actual dose

TABLE 2

2,3,7,8-CHLORINE SUBSTITUTED DIBENZODIOXINS AND DIBENZOFURANS

Isomer Group	Total Compounds in Isomer Group	Number of Compounds in Isomer Group with 2,3,7,8 Substitution	Specific Isomers
CDDs:			
Tetra-	22	1	2,3,7,8-tetraCDD
Penta-	14	1	1,2,3,7,8-pentaCDD
Hexa-	10	3	1,2,3,4,7,8-hexaCDD
			1,2,3,6,7,8-hexaCDD
			1,2,3,7,8,9-hexaCDD
Hept-	2	1	1,2,3,4,6,7,8-heptaCDD
Octa-	1	1	1,2,3,4,6,7,8,9-octaCDD
Total tetra through octaCDD compounds	49	7	
CDFs			
Tetra-	38	1	2,3,7,8-tetraCDF
Penta-	28	2	1,2,3,7,8-pentaCDF
			2,3,4,7,8-pentaCDF
Hexa-	16	4	1,2,3,4,7,8-hexaCDF
			1,2,3,6,7,8-hexaCDF
			1,2,3,7,8,9-hexaCDF
			2,3,4,6,7,8-hexaCDF
Hepta-	4	2	1,2,3,4,6,7,8-heptaCDF
			1,2,3,4,7,8,9-heptaCDF
Octa-	1	1	1,2,3,4,6,7,8,9-octaCDF
Total tetra through octaCDF compounds	87	10	

Studies whose purpose has been to determine the mutagenic potential of CDDs and CDFs have produced conflicting results. One of the reasons for this, at least for 2,3,7,8-tetraCDD, is that its high toxicity may preclude demonstration of a mutagenic response.

Carcinogenicity

Both 2,3,7,8-tetraCDD and a mixture of two hexaCDDs are potent animal carcinogens, as noted. At this time, although many people have been regularly exposed to CDD-contaminated formulations, there is little conclusive evidence linking CDD to human cancers. The difference between laboratory and human observations is surprising.

Public Law 96-151, enacted in December 1979, mandated the U. S. Veterans Administration to perform a comprehensive review and analysis of the world literature on Agent Orange and other phenoxy herbicides. Output from the original task has continued as a series of publications with Volumes IX and X being published in May 1987. These latest analyses show some associations between exposure to phenoxy herbicides, which may or may not have contained dioxins, and adverse human health impacts. However, the cited studies are noted to have shortcomings which "limit their usefulness as evidence of a cause-and-effect relationship." These include negative findings in observations made by other researchers and lack of ability to correlate effect with known exposure dose, or even to determine conclusively that all affected persons were exposed to the herbicide. One recent observation that needs further study is a statistically significant excess of non-Hodgkin's lymphoma in U. S. Marine Corps veterans who served in Vietnam compared to those who did not serve in Vietnam.

5. CRITERIA, STANDARDS, AND REGULATIONS

In the United States and Canada, criteria have been developed for certain chlorinated dibenzodioxins but not chlorinated dibenzofurans. The CDDs identified are 2,3,7,8-tetraCDD and "hexaCDD". The only agency to have adopted criteria as legally enforceable standards is the New York State Department of Environmental Conservation. The standards are for 2,3,7,8-tetraCDD: (a) 1 part per quadrillion in ambient water (10^{-6} ug/l); and (b) 35 parts per quadrillion in ground water (3.5×10^{-5} ug/l). The former is lower because of potential for bioaccumulation by aquatic organisms.

EPA has developed several criteria for 2,3,7,8-tetraCDD including those for the following: (1) ambient water for drinking purposes only (0.2 parts per quadrillion); (2) ambient water based on

Phototransformation

CDDs and CDFs resist sunlight-induced breakdown when they are present in water and on dry surfaces such as soil, wood, and glass (i.e., solid-phase surfaces). This resistance is increased with increasing number of chlorine atoms in the molecule. When chlorine atoms are lost under solid-phase conditions, those in the most toxic 2,3,7,8-substituted positions appear to be preferentially retained. This relative stability contrasts with the instability demonstrated in laboratory experiments. In these, CDDs and CDFs were dissolved in organic solvents, which enhance breakdown, and were irradiated with ultraviolet light. These conditions promote transformation to less toxic CDDs and CDFs. In the field, if organic solvents are present, they would enhance the transport of CDDs and CDFs through the soil out of range of sunlight effects. This is a current explanation for the finding of CDDs in California and Florida ground water. Until recently, CDDs and CDFs were thought to be immobile in soil, tightly bound to soil particles due to their low water solubility, and therefore not a threat to ground water.

Microbial Degradation

Unlike the sometimes marked degradative effect that microorganisms have on many compounds, CDDs either resist transformation or are only slowly degraded by microorganisms.

Sometimes, transformation compounds cannot be identified and therefore their toxicities cannot be estimated. One fungal species has been shown to degrade 2,3,7,8-tetraCDD in a nitrogen-limited culture. The usefulness of this fungus to transform CDD contaminated soil awaits evaluation. Literature on microorganism effects on CDFs is lacking, but these compounds probably show similar resistance to transformation.

Volatilization

The U.S. EPA has recently noted that volatilization is a likely fate for CDDs in aquatic environments. This contrasts with an earlier conclusion that volatilization probably was not an important process. It is consistent with a 1981 evaluation by the National Research Council of Canada: In simulating the fate of 2,3,7,8-tetraCDD in two model aquatic ecosystems, 100 percent was estimated to be lost through volatilization and none to photolysis or microbial degradation. The Research Council concluded in 1984 that despite a lack of data for CDFs, but by inference from CDD data, volatilization could play a role in environmental distribution for this class of compounds.

6. WOOD TREATMENT PRACTICES AND CALIFORNIA SITE CONTAMINATION

Chlorophenols such as pentachlorophenol (PCP), tetrachlorophenol (TCP), and their potassium and sodium salts, creosote, coal tars, and copper arsenate compounds have been used routinely at sawmills and wood treatment facilities in California. Wood is typically treated by immersing it in tanks containing the preservative solution, by spraying, or by forcing the solution under pressure into the wood to saturate the cells more fully for a longer lasting protection. Over time there is an accumulation of chemical residuals in sediments and sludge of the treatment systems. Often the treating, sorting, and drying areas become contaminated by the preservative solution.

Chlorophenols are recognized to contain CDDs and CDFs. The used preservative solution, including accumulated sediment and sludge, and contaminated soil, also contain CDDs and CDFs. Currently these wastes must be either stored on-site or disposed of outside of California because CDD and CDF-containing wastes are no longer accepted at California landfills. A nationwide ban on land-filling of dioxin-containing wastes goes into effect November 9, 1988.

On-site methods of disposal have been attempted; none are effective. These include burning in a teepee burner which, because of relatively low temperature burning, not only does not destroy CDDs and CDFs, but also produces them from precursor chlorophenol compounds. In addition, this procedure releases them to the environment adsorbed to the soot. Burial of wastes on-site also has been a common practice. As a temporary measure, on-site storage and containment of these materials in drums has been recommended as an interim disposal practice, but a long-term solution is still needed.

Three examples of California contamination occurring as a result of wood treatment operations are described. Each of these is in a different stage of the evaluation and cleanup process. They are representative of several additional sites in the state which are awaiting further investigation.

Oroville Wood Treatment Site: A 200-acre wood treatment facility near Oroville, Butte County, has been associated with the lumber industry since about 1920. Both PCP and creosote have been found in soil and ground water, both on and off-site. PCP in concentrations of up to 15,000 ppb has been detected in ground water below the site. (The California Department of Health Services Drinking Water Action Level for PCP is 30 ppb.) The depth to water is approximately 30 feet. A plume of PCP in concentrations up to 2000 ppb has been detected at least two miles south of the site. The depth to water in this area is

adsorbed and absorbed CDDs and CDFs are both consumed with the food. With respect to terrestrial plants, EPA has recently concluded that 2,3,7,8-tetraCDD present in contaminated soil is "not likely" to concentrate in them. If true, plants would not be effective scavengers of CDDs and CDFs in soil, a use which has been suggested for on-site treatment of CDD and CDF-contaminated soil. Reported bioconcentration of CDDs by terrestrial plants may be due to contamination of leaf and plant surfaces by CDDs in dust and soil particles.

3. AQUATIC TOXICOLOGY

Two striking aspects of 2,3,7,8-tetraCDD toxicity to aquatic life are the (1) delayed toxic effects after brief periods of exposure; and (2) low concentrations which cause toxic reactions. Frequently, toxicity is not seen in the standard short-term, 96-hour acute test. Statistically significant adverse effects have been delayed for periods ranging from five to over 100 days after exposure to this chemical. Growth retardation is the most common effect reported for 2,3,7,8-tetraCDD. Other effects include fin necrosis, loss or underdevelopment of caudal fins, edema, liver necrosis, and hemorrhaging.

Toxic effects have been reported at water concentrations as low as 0.1 parts per trillion (ppt) 2,3,7,8-tetraCDD. The lowest acute LC_{50} value of 5.6 ppt for coho salmon is one order of magnitude lower than for two of the most toxic chemicals to aquatic life, endosulfan and toxaphene. (LC_{50} refers to the concentration of a chemical which kills 50 percent of a test population within a specified time period.)

Due to the delayed lethality normally found in 2,3,7,8-tetraCDD bioassays, the expression of LC_{50} for a 96-hour exposure is not a meaningful indicator of 2,3,7,8-tetraCDD toxicity. As a result, the literature concerning 2,3,7,8-tetraCDD describes modified LC_{50}s indicating mortality at some given time after the exposure period. There is no agreement on a standardized post exposure observation period for the calculation of LC_{50}.

Most toxicity studies with CDDs have focused on 2,3,7,8-tetraCDD. They have generally been short-term 96-hour exposures, and have been "static" or "static renewal" bioassays. The water and test chemical were either not renewed for the test period, or were renewed periodically as a batch replacement. Studying the toxicity of highly water insoluble compounds such as CDDs under static testing conditions can present difficulties. For example, a compound will tend to migrate out of the aqueous test solution and adsorb onto solid surfaces such as the test container, test organisms, or particulate debris. The adsorbed test chemical may not be available to the test organisms.

influent, effluent, and sludge with one exception: CDDs and CDFs were not detected in the plant effluent. Water from the deep aquifer is used by the City of Visalia for drinking water. Sludge from the treatment plant has been used as a soil amendment by farms and residents. CDFs have been detected in soil. In 1985 a pretreatment system was installed at the site to remove ground water contaminants before water transfer to the treatment plant.

7. CALIFORNIA WATER RESOURCES CONTROL BOARD STUDY

The study reported here originally was based on potential pentachlorophenol contamination of the environment. The focus was on its use by sawmills and wood treatment plants. Samples taken for analysis included aquatic invertebrates and fish, treatment site runoff, ground water, and soil. At that time, State Board staff considered that environmental contamination by chlorinated dibenzodioxins and dibenzofurans might be of equal significance. (They were known to be contaminants of chlorophenol formulations.) To test this hypothesis, 13 samples were taken from five sawmills and one wood treatment plant (Table 3, Section A). Sample types and numbers were as follows: soil (4), sludge (4), dip tank liquid (2), and commercial chlorophenol formulations (3). Analyses detected significant CDD and CDF concentrations. A decision was made to base the study on CDD and CDF presence in areas of sawmills and wood treatment plants. Chlorophenols would become the subject of another survey.

This initial work showed that tetra-, penta-, hexa-, hepta-, and octaCDDs and CDFs were present in all 13 samples, with one exception; tetraCDDs were detected only in wet and dry sludge samples from one sawmill and in one pentachlorophenate commercial product. The commercial chlorophenol and chlorophenate products were found to contain both tetra-chlorophenol and pentachlorophenol.

Analyses at this stage identified CDDs and CDFs in terms of "isomer groups", e.g., "tetraCDD", "heptaCDF". The analyses did not identify specific CDDs and CDFs, e.g., 2,3,7,8-tetraCDD. Determination of the exact position of the chlorine atoms requires a rigorous analytical procedure. As noted earlier, a total of 210 individual CDDs and CDFs possibly can occur.

After CDD and CDF presence was firmly established in the 13 samples, a decision was made to concentrate future work on the 15 CDDs and CDFs that were toxicologically most significant, i.e., the tetra, penta, hexa, and hepta-chlorinated compounds

higher concentrations tested caused mortality within 28 days, the toxic effect of lower concentrations was not manifested until later. During the 28-day depuration period, mortality continued and there was no observed recovery in clean water.

The same study also reported bioconcentration factors of 39,000 for 2,3,7,8-tetraCDD and 6,049 for 2,3,7,8-tetraCDF. This newly published study concluded that 2,3,7,8-tetraCDD is more than 10,000 times as toxic to fish as the insecticides endrin or toxaphene and that 2,3,7,8-tetraCDF is roughly 1,000 times as toxic.

4. MAMMALIAN TOXICOLOGY

Absorption, Tissue Distribution, Metabolism, and Half-Lives

Both CDDs and CDFs are absorbed and concentrated by laboratory animals and humans. Up to 90 percent of the chemicals will be absorbed if they are present in food. Approximately 40 percent can be absorbed after skin application to laboratory animals.

The half-life of 2,3,7,8-tetraCDD in a 42 year old human volunteer was estimated to be 5.8 years. This is longer than the half-life of about one year for the same compound estimated for monkeys. It contrasts with 10 to 40 day half-lives measured in several small laboratory animals. Based on blood sample analyses, a half-life of greater than one year was calculated for 2,3,4,7,8-pentaCDF and 1,2,3,4,7,8-hexaCDF compounds in humans. These people had ingested rice oil contaminated with these and other CDFs in Taiwan. In Japan, following a similar incident, the same pentaCDF could still be detected in human blood 11 years after exposure.

Studies with 2,3,7,8-tetraCDD contaminated soil show that ingested soil can influence toxicity. Soil from a Times Beach, Missouri, area which was contaminated with waste oil containing CDDs and CDFs, produced a variety of adverse effects, including acute toxicity in laboratory studies. In contrast, contaminated soil from a 2,4,5-T and 2,4-D formulation site in New Jersey produced no toxicity in laboratory animals. Bioavailability of the chemicals, including CDDs and CDFs, appears to account for the difference between these two observations. This was estimated to range from 0.5 to 21 percent for the New Jersey soil and 25 to 85 percent for the Times Beach soil. Bioavailability refers to the amount which is expected to be absorbed into the animal's bloodstream and not tightly bound to the soil particles which would be eliminated as waste.

which have four of the chlorine atoms located in the 2,3,7, and 8 positions (Table 2). Three of the target compounds are potent carcinogens to laboratory animals: 2,3,7,8-tetraCDD and a mixture of 1,2,3,6,7,8- and 1,2,3,7,8,9-hexaCDD.

In order to estimate the concentration of these 15 compounds in complex mixtures, which also include many non-2,3,7,8-substituted CDDs and CDFs, three methods can be followed. The first two are fairly straightforward. They require little more time than that to determine isomer group concentrations. One of these methods assumes that all tetra through heptaCDDs and CDFs are chlorinated at positions 2,3,7, and 8. This procedure could greatly overestimate the significance of the 2,3,7,8-substituted compounds since there may be a far greater number of non-2,3,7,8-substituted compounds present. The second method assumes that all compounds within an isomer group are present in equal numbers; e.g., 2,3,7,8-tetraCDD is one of 22 possible compounds in the tetraCDD isomer group, and its concentration would be 1/22 of the total tetraCDD concentration detected. While simple in concept, this procedure could significantly underestimate or overestimate the toxicity of a CDD mixture, depending whether or not 2,3,7,8-tetraCDD was present. The third method identifies each 2,3,7,8 CDD and CDF in a potential mixture of 210 CDDs and CDFs and numerous other interferences. This approach represents state-of-the-art analytical chemistry for CDDs and CDFs. It was the course chosen for the study reported here.

The work proceeded in two phases. Phase 1 was directed at analyzing some of the previously collected samples which were shown to contain high concentrations of CDD and CDF isomer groups. Phase 2 was initiated with additional samples once 2,3,7,8-substituted CDDs and CDFs were identified in Phase 1. All samples analyzed are described in Table 3.

A brief summary of the analytical results follows. All data are described in detail in the accompanying report appendices.

CDDs: 2,3,7,8-Chlorinated compounds from all four target isomer groups (tetra through hepta) were detected in all 12 samples analyzed, with the following exceptions: 2,3,7,8-tetraCDD was detected in only one sample and 2,3,7,8-pentaCDDs were detected in five of 12 samples.

CDFs: 2,3,7,8-Chlorinated compounds from all target isomer groups were detected in the 12 samples analyzed, with the following exceptions: tetraCDFs were found in 9 of 12 samples, with pentaCDFs and hexaCDFs in 10 of 12.

received could not be determined. The most common human effects attributed to 2,3,7,8-tetraCDD exposures include chloracne, liver abnormalities, hematologic disorders, porphyria, and hyperpigmentation disorders. Also reported have been peripheral and central neurological disorders, lethargy and sensory impairment. Chloracne is characterized by comedones and cysts. These may subside within a few months or persist for years, with some cases reported lasting up to 15 years after exposure.

Other human exposure sources to 2,3,7,8-tetraCDD include (1) dirt roads in Missouri sprayed with waste oil containing 2,3,7,8-tetraCDD, and (2) Agent Orange, the herbicide used in Vietnam also contaminated with this compound. The 2,3,7,8-tetraCDD concentrations in the Missouri soil ranged from 39 to 2200 ppb. Persons exposed to this material had lived in the area from one to five years during the period of contamination. Signs of altered liver function included lower serum bilirubin and elevated urinary uroporphyrin concentrations. However, these measurements were considered to be "subclinical"; i.e., not significantly differing from the normal.

The Agent Orange exposure is discussed in the section "Carcinogenicity" below.

The Japanese rice oil contaminated with 1,000 ppm PCBs and 5 ppm CDFs, which included 0.45 ppm 2,3,7,8-tetraCDF, produced the following toxic effects in humans, collectively known as "Yusho": pigmentation disorders, chloracne, eye discharge, swelling of upper eyelids, distinctive hair follicles, and neurological disturbances.

Teratogenicity and Reproduction

2,3,7,8-TetraCDD is a teratogen to laboratory animals. Cleft palate is the most common malformation observed in mice. Kidney defects are also common as well as embryo toxicity. In rats, teratogenic effects include subcutaneous edema, hemorrhage in the gastrointestinal tract, kidney malformation, cleft palate, and vertebral defects. In monkeys there are insufficient data to clearly define a teratogenic response, although fetotoxicity has been observed. Studies of humans exposed to 2,3,7,8-tetraCDD in the chemical industry, during the Vietnam war and in forestry operations, have not been able to show a teratogenic or other adverse effect on reproduction. The animal data conclusively demonstrate that 2,3,7,8-tetraCDD is teratogenic and fetotoxic at low levels of exposure. They indicate a need to determine more carefully the potential for adverse human reproductive effects.

TABLE 4

SUMMARY OF 2,3,7,8-SUBSTITUTED CDD AND CDF CONCENTRATIONS IN TWELVE COMPOUND-SPECIFIC ANALYSES (TETRA, PENTA, HEXA, AND HEPTA ISOMER GROUPS) - ppb[1/]

Sample	2,3,7,8-Tetra-CDD	2,3,7,8-Tetra-CDF	All 2,3,7,8-Chlorinated CDDs[2/]	All 2,3,7,8-Chlorinated CDFs[2/]	Total 2,3,7,8-CDDs & CDFs[2/]
Commercial Na-PCP, Sawmill A	0	201	34,751	6,540	41,291
Commercial K-TetraCP, Sawmill C	0	200	1,197	1,148	2,345
Sawmill Dip Tanks					
Sawmill A sludge	0	15	25,305	3,333	28,638
Sawmill B wet sludge	0	17	2,332	485	2,817
Sawmill B dry sludge	9.7	95	15,411	2,177	17,588
Sawmill C center sludge	0	54	1,092	560	1,652
Sawmill C corner sludge	0[3/]	65	1,161	574	1,735
Sawmill C liquid	0	2.0	18	26	44
Wood Treatment Plant-					
PCP "Bloom"	0	4.4	24,183	10,712	34,895
Recycled "Commercial"	0	0	6,715	726	7,441
Soil at Retort Mouth	0	0	1,618	169	1,887
Sump Liquid	0	0	8,684	69	8,753

1/ Average of samples split between two laboratories.

2/ Does not include octaCDD and octaCDF.

3/ Reported at 6.8 ppb by one laboratory but not confirmed by second.

consumption of fish and shell-fish only (0.014 parts per quadrillion); (3) ambient water based on consumption of water, fish and shellfish (0.013 parts per quadrillion); (4) total intake from all sources for humans (0.006 picograms per kilogram body weight per day); and (5) ambient air (0.03 picograms per cubic meter). Specific criteria are listed which relate to the one increased incidence of cancer per one million population risk level.

Other 2,3,7,8-tetraCDD criteria have been developed by the following agencies: (1) Michigan Department of Public Health (10 ppt in fish); (2) California Air Resources Board and Department of Health Services (30 femtograms per cubic meter in air); (3) U. S. Centers for Disease Control (1 ppb in soil); (4) U. S. Food and Drug Administration (50 ppt in fish); and (5) Ontario Ministry of the Environment, Ontario, Canada (20 ppt total intake from all sources for humans): The U. S. Food and Drug Administration also set an additional advisory level for consumption of fish containing 25 to 49 ppt 2,3,7,8-tetraCDD. Fish with concentrations in this range should not be consumed more than twice per month.

HexaCDD criteria have been developed by the following: (1) EPA (5.5 parts per quadrillion in drinking water; 0.8 picograms per cubic meter in air; and 0.16 picograms per kilogram body weight per day for all sources in humans); (2) California Air Resources Board and Department of Health Services (1 picogram per cubic meter in air); and (3) National Research Council of Canada (13 ppt in ambient water for human consumption of fish; and 20 ppt for fish flesh).

Regulations have been developed for both CDDs and CDFs which relate primarily to treatment methods and disposal. The California Department of Health Services regulates 2,3,7,8-tetraCDD in wastes disposed to land to protect against migration to surface and ground water. EPA has developed CDD and CDF treatment standards and prohibits land disposal of certain wastes containing these compounds unless treatment standards are achieved. The designated wastes include several chemicals with which CDDs and CDFs are associated as contaminants and include tri-, tetra-, and pentachlorophenol; tetra-, penta-, and hexachlorobenzene, and 2,4,5-T. They also include residues resulting from incineration or thermal treatment of soil contaminated with certain EPA-designated hazardous wastes. In addition, EPA regulations require registrants of pentachlorophenol to reduce the concentration of hexaCDD in three phases. By February 2, 1989, the maximum batch hexaCDD concentration allowed will be 4 ppm, with a maximum average of 2 ppm; this is a decrease from the present allowable maximum batch concentration of 15 ppm.

TABLE 5

TOXIC EQUIVALENCY FACTORS FOR 2,3,7,8-CHLORINATED DIBENZODIOXINS AND DIBENZOFURANS (SOURCE: BELLIN AND BARNES, 1986)

Compound	Toxic Equivalency Factor[2/]
CDD	
2,3,7,8-tetraCDD	1.0
1,2,3,7,8-pentaCDD	0.5
1,2,3,4,7,8-hexaCDD	0.04
1,2,3,6,7,8-hexaCDD	0.04
1,2,3,7,8,9-hexaCDD	0.04
1,2,3,4,6,7,8-heptaCDD	0.001
CDF	
2,3,7,8-tetraCDF	0.1
1,2,3,7,8-pentaCDF	0.1
2,3,4,7,8-pentaCDF	0.1
1,2,3,4,7,8-hexaCDF	0.01
1,2,3,6,7,8-hexaCDF	0.01
1,2,3,7,8,9-hexaCDF	0.01
2,3,4,6,7,8-hexaCDF	0.01
1,2,3,4,6,7,8-heptaCDF	0.001
1,2,3,4,7,8,9-heptaCDF	0.001

1/ Bellin, J., and D. Barnes, 1986. Interim Procedures for Estimating Risks Associated with Exposure to Mixtures of Chlorinated Dibenzodioxins and Diobenzofurans (CDDs and CDFs). Risk Assessment Forum, U. S. Environmental Protection Agency EPA/625/3-87/012. Washington, DC.

2/ Toxic Equivalency Factors are based on carcinogenicity and other toxicity data relative to that for 2,3,7,8-tetrachlorodibenzodioxin (2,3,7,8-tetraCDD).

90 to 120 feet. Approximately 30 domestic wells have been found contaminated with PCP. No CDDs or CDFs have been detected. Residents have complained of various adverse health effects. A comprehensive study is underway to define the extent of PCP contamination.

Selma Wood Treatment Site: An 18-acre wood treatment facility has been in operation since approximately 1936 near Selma, Fresno County. As with similar facilities, a number of preservative chemicals have been used here including chromated copper arsenate and pentachlorophenol dissolved in a variety of solvents. Wastes were discharged into dry wells, into an unlined pond, as runoff into drainage ditches, to open ground, and into a sludge pit. PCP has been detected on-site in surface and ground water and in soil. Surface water concentrations have ranged from 0.24 to 2.3 ppm. The PCP ground water concentration was determined to be 2 ppb. The depth to water is approximately 30 feet. All tetra-through-octaCDD and CDF isomer groups, except tetraCDD, were detected in soil.

Off-site migration may have occurred since the vertical and horizontal extent of soil and ground water contamination has not been defined. EPA is currently conducting a sampling program to clarify this uncertainty.

Visalia Wood Treatment Site: This facility, in Tulare County, had used PCP for electrical pole treatment from 1968 to 1980, when operations ceased. Ground water contamination was detected in 1973 and has been followed since then. Hexa-, hepta-, and octaCDDs and CDFs, and PCP have been detected in shallow and deep aquifers. These and pentaCDFs also were detected in soil. There were no pentaCDDs detected in soil. PCP was detected in monitoring wells 600 feet to the south of the site at concentrations ranging up to 37 ppm and 1600 feet to the southwest at concentrations up to 2 ppm. Creosote was found in these samples. Additional monitoring wells were constructed in 1984 and soil cores taken during this work were analyzed to provide information on the vertical distribution of PCP, creosote, CDDs, and CDFs.

Ground water has been pumped from the shallow aquifer to the City of Visalia wastewater treatment plant since 1975. The purpose has been to reduce contaminant concentrations and prevent further migration away from the site. Additionally, a bentonite-cement slurry wall has been built below the surface to inhibit down-gradient movement of the contaminants. The barrier surrounds the shallow aquifer beneath the site and extends from the surface to its lower boundary. PCP, creosote, CDDs, and CDFs were detected in both aquifers whose waters were discharged to the treatment plant. All of these compounds were detected also in plant

TABLE 6

TOTAL RELATIVE TOXICITY CONCENTRATIONS (ppb) OF 2,3,7,8-CHLORINATED DIBENZODIOXINS AND DIBENZOFURANS: A COMPARISON BASED ON THREE METHODS[1/]

	Method 1	Method 2	Method 3
SAMPLE	TEF=1[2/]	CDHS 1986[3/]	Bellin and Barnes, EPA 1986[4/]
Commercial Na-PCP			
Sawmill A	41,291	2,055	289.5
Commercial K-tetraCP			
Sawmill C	2,345	463	72.8
Sawmill Dip Tanks			
Sawmill A sludge	28,638	1,184	139.1
Sawmill B wet sludge	2,817	173	32.0
Sawmill B dry sludge	17,588	1,094	329.6
Sawmill C center of tank sludge	1,652	216	27.0
Sawmill C corner of tank sludge	1,735	218	27.9
Sawmill C liquid	44	8.4	0.8
Wood Treatment Plant-PCP			
"Bloom"	34,895	1,120	100.5
Recycled "Commercial"	7,441	223	11.3
Soil at Retort Mouth	1,887	64	5.6
Sump Liquid	8,753	274	9.8

1/ OctaCDD and octaCDF were not considered in the calculations due to estimated low toxicity.

2/ Toxic Equivalency Factor = 1 for each 2,3,7,8-chlorinated CDD and CDF.

3/ California Department of Health Services, 1986; Relative potency of 2,3,7,8-tetra- and pentaCDDs and CDFs = 2,3,7,8-tetraCDD; and 2,3,7,8-hexa- and heptaCDDs and CDFs = 2,3,7,8-hexaCDD (or 0.03 2,3,7,8-tetraCDD).

4/ Bellin, J. and D. Barnes. 1986. Interim Procedures for Estimating Risks Associated with Exposure to Mixtures of Chlorinated Dibenzodioxins and Dibenzofurans (CDDs and CDFs). Risk Assessment Forum, U. S. Environmental Protection Agency EPA/625/3-87/012. Washington, DC.

TABLE 3

CALIFORNIA WATER RESOURCES CONTROL BOARD
CHLORINATED DIBENZODIOXIN AND DIBENZOFURAN STUDY

A. Preliminary Screening: Isomer Group Analyses

1. 5 Sawmills and 1 Wood Treatment Plant: 13 samples as indicated:

   a. Soil (4)
   b. Sludge (4)
   c. Dip tank liquid (2)
   d. Commercial formulations (3)

B. Phase I: Compound Specific Analyses

1. Sawmill A (Trinity County): 2 samples

   a. Commercial sodium pentachlorophenate
   b. Dip tank sludge

2. Sawmill B (Glenn County): 2 samples

   a. Wet dip tank sludge
   b. Dry mix tank sludge

C. Phase II: Compound Specific Analyses

1. Sawmill C (Humboldt County): 4 samples

   a. Commercial potassium tetrachlorophenate
   b. Dip tank liquid
   c. Dip tank sludge (2 samples)

2. Wood Treatment Plant (San Joaquin County): 4 samples

   a. "Bloom"
   b. "Commercial"--recycled treatment material
   c. Soil at retort
   d. Sump liquid

CHAPTER 1: INTRODUCTION

Why are polychlorinated dibenzo-p-dioxins (CDDs) and dibenzofurans (CDFs) important? The best known and most studied of the CDDs is the chemical 2,3,7,8-tetrachlorodibenzo-p-dioxin, commonly called "dioxin". In ranking the potency of 55 suspected human carcinogens, the U.S. EPA (1985b) listed "dioxin" as the most potent -- 50 million times more potent than trichlorethylene (TCE) or vinyl chloride. This CDD compound also is highly toxic in a single dose to certain animal species. In a single feeding of one part "dioxin" to one billion parts body weight, half of the guinea pigs dosed will die. However, unlike a lethal dose from many other highly toxic chemicals, death is delayed from 5 to 45 days after exposure occurs. In addition to 2,3,7,8-tetrachlorodibenzo-p-dioxin, several other CDDs and CDFs are of probable toxicological concern.

BACKGROUND

The California Water Resources Control Board's (State Board) investigation of chlorinated dibenzo-p-dioxins and dibenzofurans originated with a study of pentachlorophenol (PCP). This compound is a major industrial chemical and biocide used worldwide. In California, PCP has been used extensively for wood preservation at lumber mills and wood treatment plants. Typically, a water soluble form of PCP is used at sawmills for surface protection against fungal staining of lumber. In contrast, wood treatment plants inject insoluble PCP under pressure for long-term protection of materials such as poles and posts. Most of these facilities are located in two areas of the state, the northwest and the central valley. Investigations by the Regional Water Quality Control Boards and other agencies have documented a number of effects on California's environment. These include fish kills; contaminated soil, surface water and ground water; accumulation in marine sediments and organisms; and incidents of worker exposure. A few California studies have also detected CDDs and CDFs in both commercial PCP and PCP-contaminated soil.

In the past five years, conditions at sawmills have noticeably improved. Some mills have converted to systems that completely contain and recycle wood preservative chemicals on site, preventing environmental contamination. In particular, the "unit dip" tank has been successful. In a unit dip operation, a below ground rectangular tank is filled with a wood preservation solution (typically the soluble form of pentachlorophenol is diluted 1:100 parts water). Sawn lumber is bundled together, immersed in the tank, then allowed to dry in a covered building sloped such that drippage drains back into the dip tank. A side

The concentration of the 2,3,7,8-substituted compounds was calculated also as a percentage of the total CDD or CDF concentration for each isomer group. Depending on the sample and the isomer group, the proportion of the 2,3,7,8 compounds ranged from a few percent to greater than 80 percent of the total concentration of the respective isomer group. This finding was based on analysis of the environmental samples and the two commercial chlorophenate products.

All samples except for one dip tank solution contained at least 1,000 ppb total tetra-through-hepta 2,3,7,8-chlorinated CDDs and CDFs (Table 4; Total 2,3,7,8 CDDs and CDFs). The total concentration of tetra-through-hepta 2,3,7,8-chlorinated CDDs and CDFs ranged between 44 and 41,000 ppb. The concentrations of 2,3,7,8-tetraCDD and CDF are given separately because of the high toxicity of the former, and of the latter by analogy. The presence of 2,3,7,8-tetraCDF is particularly significant because of its close structural resemblance to 2,3,7,8-tetraCDD. The table shows that 2,3,7,8-tetraCDF was present in all 8 samples taken at sawmills, up to concentrations of 200 ppb.

The study data also show that the following 2,3,7,8-chlorinated CDD and CDF compounds are most likely to be found as a result of tetrachlorophenol and pentachlorophenol use at sawmills and wood treatment plants.

1,2,3,6,7,8-hexaCDD
2,3,7,8-tetraCDF
1,2,3,7,8-pentaCDF
2,3,4,7,8-pentaCDF
1,2,3,6,7,8-hexaCDF

Although the data are complex, a brief overview of analyses of these 12 samples indicates the following: 2,3,7,8-chlorinated CDDs and CDFs are present as contaminants at sawmills and wood treatment plants, often at significant concentrations.

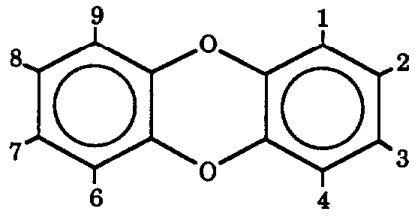
8. HAZARD EVALUATION

Compound Detection

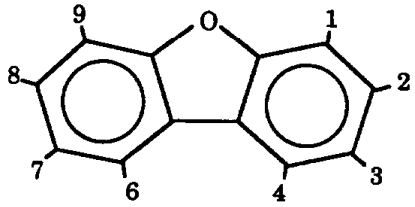
As noted, a major assumption was made that most of the toxicity in CDD and CDF mixtures is contributed by the 2,3,7,8-chlorinated compounds. Laboratories in the United States and Sweden participating in the State Board Study obtained analytical standards for the 15 most toxic 2,3,7,8-CDDs and CDFs. Often these had to be synthesized since they were not commercially available. Analytical procedures were developed and refined for their detection. When detected in a sample, a concentration for each 2,3,7,8-chlorinated CDD and CDF was determined for each of

FIGURE 1.1

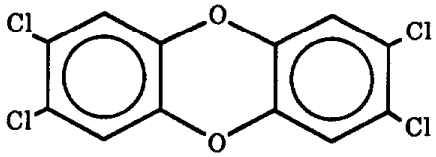
CHEMICAL STRUCTURES



Dibenzo-p-dioxin



Dibenzofuran



2,3,7,8-Tetrachlorodibenzo-p-dioxin
(2,3,7,8-TetraCDD)
("Dioxin")

the 12 samples. For each of the samples, the relative proportion of the 2,3,7,8-chlorinated CDDs and CDFs in each isomer group also was calculated.

Toxicity Evaluation

Toxicity information was available for only a few of the 2,3,7,8-chlorinated compounds. In order to overcome this deficiency, three methods were considered to determine total sample toxicity based on toxicity of the individual 2,3,7,8-compounds.

1. The simplest approach is to assign the same "toxic equivalency factor" to each 2,3,7,8-chlorinated tetra-, penta-, hexa-, and heptaCDD and CDF, i.e., assume they are all equally toxic. The toxic equivalency factor is multiplied by the concentration of each compound detected to yield a "relative toxicity concentration." All products are added together to estimate a "total relative toxicity concentration" for all CDDs and CDFs in each sample.

   This approach does not take into consideration the different toxicities of individual compounds. It can be justified on the basis of limited toxicity information for most of the 2,3,7,8-substituted compounds, taking into account that toxicity generally was high where it has been measured.

2. The California Department of Health Services currently favors an approach which is based solely on data provided by carcinogenicity bioassays. Only two toxic equivalency factors can be estimated with this scenario because only 2,3,7,8-tetraCDD and a mixture of two 2,3,7,8-chlorinated hexaCDD compounds have been tested for carcinogenicity. With this method, all other CDDs and CDFs are assigned one or the other of the two factors. As with the first approach just described, each factor is multiplied by the appropriate compound concentration to estimate a relative toxicity concentration for each compound. The products also are added to estimate a total relative toxicity concentration for all CDDs and CDFs.

3. The U.S. Environmental Protection Agency has developed toxic equivalency factors for the 2,3,7,8-chlorinated CDDs and CDFs by taking into consideration both carcinogenicity information and other toxic effects data, such as those relating to reproductive effects. These equivalency factors are listed in Table 5. EPA also considers toxicity of non-2,3,7,8-chlorinated CDDs and CDFs and assigns them factors. These are one to three orders of magnitude less than those for the respective chlorinated compounds. Relative toxicity concentrations and total toxicities are estimated using the same steps described for the first two approaches.

TABLE 1.1

CDD AND CDF ISOMER GROUPS, ISOMERS, AND CONGENERS

Isomer Group	Number of Isomers in Isomer Group
CDDs	
1. Monochlorodibenzodioxin (monoCDD)	2
2. Dichlorodibenzodioxin (diCDD)	10
3. Trichlorodibenzodioxin (triCDD)	14
4. Tetrachlorodibenzodioxin (tetraCDD)	22
5. Pentachlorodibenzodioxin (pentaCDD)	14
6. Hexachlorodibenzodioxin (hexaCDD)	10
7. Heptachlorodibenzodioxin (heptaCDD)	2
8. Octachlorodibenzodioxin (octaCDD)	1
TOTAL CDD CONGENERS	75
CDFs	
1. Monochlorodibenzofuran (monoCDF)	4
2. Dichlorodibenzofuran (diCDF)	16
3. Trichlorodibenzofuran (triCDF)	28
4. Tetrachlorodibenzofuran (tetraCDF)	38
5. Pentachlorodibenzofuran (pentaCDF)	28
6. Hexachlorodibenzofuran (hexaCDF)	16
7. Heptachlorodibenzofuran (heptaCDF)	4
8. Octachlorodibenzofuran (octaCDF)	1
TOTAL CDF CONGENERS	135

by splitting samples between laboratories; and (2) determine if 2,3,7,8 CDD and CDF congeners were present in sawmill residues. This latter approach, referred to as congener-specific analysis, is difficult and represents state-of-the-art analytical chemistry. This report describes the results of subsequent 2,3,7,8 congener-specific analysis commissioned by State Board staff, as well as the results of the earlier isomer group analyses.

A comparison of the total relative toxicity concentrations estimated by the three methods for each of the 12 samples, shows a difference of three orders of magnitude between them (Table 6). The most conservative, i.e. highest, concentrations are based on the sum of all tetra through hepta 2,3,7,8-chlorinated CDDs and CDFs which have been given the same toxic equivalency factor (Method 1). These concentrations were 5 to 30 times greater than those calculated by the California Department of Health Services procedure (Method 2). The latter, in turn, were 3 to 30 times higher than those calculated by the EPA approach (Method 3). For example, the total relative toxicity concentrations calculated by the three methods for the "Sawmill B" dry-sludge sample were 17,588 ppb, 1094 ppb, and 330 ppb, respectively.

The authors of this report recommend that, until more 2,3,7,8-chlorinated compound-specific toxicity information is available, the EPA procedure be used to estimate the total relative toxicity concentrations of CDD and CDF mixtures. This method, unlike the previous two, takes into account all available toxicity information for the various CDD and CDF compounds.

Comparisons of relative toxicity concentrations also were made between CDD and CDF "isomer groups" in each sample for the 2,3,7,8-substituted compounds. In one sawmill dip-tank sludge sample, the compounds contributing the most relative toxicity, based on the EPA method, were the pentaCDFs (38 percent); the hexaCDDs (32 percent); and 2,3,7,8-tetraCDF (23 percent). These figures are based on a total sample relative toxicity concentration for CDDs and CDFs of 27.9 ppb (Sawmill C, Table 6).

The relative toxicity concentration of all 2,3,7,8-substituted CDFs in the same sample was approximately twice that of the CDDs. The estimated relative toxicity concentration for these CDFs was 18 ppb and for CDDs, 9.9 ppb.

Future Sample Toxicity Evaluation

The authors recommend a simplified approach to estimating total CDD and CDF toxicity of similar samples in future analyses. It is based on (1) performing isomer group analyses; and (2) using the ratios of the 2,3,7,8-chlorinated compounds identified in this study, relative to isomer group concentrations. These ratios can be used to estimate relative toxicity concentrations for similar sample types, when only isomer group analyses are performed. The current data bases (12 samples) can be increased by additional compound-specific analyses by other specialist laboratories. CDD and CDF isomer group analyses can be performed by many commercial laboratories. Only a few laboratories in the United States are capable of doing the more definitive analyses on a reasonable schedule.

CHAPTER 2: ENVIRONMENTAL FATE

Despite considerable research, there are little reliable qualitative or quantitative data with which to predict the environmental fate of 2,3,7,8-tetraCDD accurately. Much less is known about other polychlorinated dibenzo-p-dioxins (CDDs) (U.S. EPA, 1985b) and even less about the polychlorinated dibenzofurans (CDFs) (NRCC, 1984). The bulk of observations in this chapter, therefore, concern 2,3,7,8-tetraCDD; inferences occasionally are made concerning the behavior of other CDDs and CDFs based on 2,3,7,8-tetraCDD behavior.

Sections of this chapter address five areas: (1) phototransformation; (2) microbial degradation; (3) volatilization; (4) persistence and movement in soil and sediment; and (5) plant uptake. Major emphasis is placed on phototransformations, as this area has been a subject of considerable research and some controversy.

PHOTOTRANSFORMATION

The subject of CDD and CDF phototransformations has received considerable attention in recent years. Until recently, it was believed that these compounds would break down to less toxic compounds in the environment. However, it is now known that many of the conditions used in laboratory studies are not necessarily representative of environmental situations. Major differences are apparent depending on whether the photoreactions occur in organic solvents ("solution phase" reactions) or on surfaces such as wood or glass ("solid phase" reactions). Under solid phase conditions, it is possible for more toxic CDDs and CDFs to be produced from less toxic cogeners.

Polychlorinated dibenzo-p-dioxins (CDDs) and dibenzofurans (CDFs) have been shown to be both formed and degraded by sunlight (NRCC, 1984; U.S. EPA, 1985b). Precursor compounds are found in commercial chlorinated phenol wood preservative formulations and include chlorinated diphenyl ethers, "predioxins" (chlorinated-2-phenoxyphenols), and tetrachlorophenol and pentachlorophenol (Figure 2.1) (Norstrom et al., 1977; Nilsson et al., 1974; Choudhry and Hutzinger, 1982; Kitunen et al., 1987). Further, highly chlorinated CDDs and CDFs may be dechlorinated photolytically to produce more toxic CDDs and CDFs (Buser, 1976; U.S. EPA, 1985b). A summary of phototransformation reactions of CDDs and CDFs is given in Table 2.1, and described in more detail as follows:

The only site-specific cleanup level that has been established for CDDs or CDFs in the United States has been 1 ppb for 2,3,7,8-tetraCDD in Times Beach, Missouri. Total relative toxicity concentrations calculated for the 12 samples in this study -- using the EPA method -- showed that 11 exceeded 1 ppb. These ranged from 5.6 to 329.6 ppb (Table 6).

The concentration for the twelfth sample was 0.8 ppb. All 12 exceeded the 1 ppb level based on the California Department of Health Services' method of calculation.

Setting a Clean Up Level. Contamination by CDD and CDF mixtures associated with chlorophenol products used at sawmills and wood treatment plants should be cleaned up following a site-specific procedure. The present study concludes that the DHS California Site Mitigation Decision Tree Manual, although complex, should be followed. The Decision Tree includes five components:

1. Preliminary risk appraisal;
2. Site assessment;
3. Risk appraisal;
4. Environmental fate and risk determination; and
5. Determination of mitigation strategy and remedial action plan selection.

The risk appraisal phase uses applied action levels (AAL) for specific media of exposure such as air, soil, water, and biota. These have been set to protect specific biological "receptors". The AALs also take into account the amount of a substance taken in by inhalation, ingestion, and adsorption, as well as other toxicological factors such as absorption, metabolism, distribution, and elimination characteristics of the medium.

The California Department of Health Services is currently reviewing a consultant's report containing proposed air and water AALs for CDDs. A CDHS report describes a strategy for developing AALs related to soil contact. Numerical AALs for CDDs in soil will be proposed by CDHS in 1988.

Characterization of CDD and CDF mixtures in samples by calculating total relative toxicity concentrations will allow an estimate of potential hazard. The options for remedial action can then be identified. At some sites, moving the material may create more of a hazard than encapsulation and on-site storage. On-site storage with material isolated from humans and the environment may be the most effective interim measure until acceptable methods of CDD and CDF destruction are available.

TABLE 2.1

SUMMARY OF PHOTOTRANSFORMATIONS OF CDDs AND CDFs

Process	Medium	Compound or Precursor	Fate or Product	Reference
Formation Reactions				
1.	water,UV	Na-PCP	octaCDD	Crosby et al., 1973
2.	wood, sunlight	PCP	octaCDD	Cull and Dobbs, 1984
3.	wood, sunlight	purified PCP	octaCDD heptaCDD hexaCDD	Lamparksi et al., 1980
4.	methanol, UV	predioxins	tetraCDD triCDDs diCDD diCDF	Nilsson et al., 1974
5.	hexane UV	PCDE (polychlori-nated diphenyl ethers)	CDFs	Norstrom et al., 1976 and 1977
6.	methanol, UV	PCB (tetra-chloro-PCB)	CDF (diCDF)	Choudhry and and Hutzinger, 1982
Photolysis				
A. Solution Phase	organic solvents, UV or sunlight	higher CDDs or CDFs	lower CDDs or CDFs (preferential lateral chlorine removal)	Buser and Rappe, 1978
1.	benzene-hexane, UV	octaCDD	1,2,3,4,6, 7,9-heptaCDD (major product)	Buser, 1976

effect of this "best management practice" is accumulation of sawdust and dirt on the tank bottom that forms a sludge. Eventually, the sludge becomes deep enough to interfere with dip operations and must be removed. An examination of two Swedish sawmills that used chlorinated phenols as wood preservatives noted that these sludges became "remarkedly enriched" in chlorinated dibenzofurans (Levin et al., 1976). Levels of total CDFs as high as 700 ppm were detected. Upon learning of the findings in Sweden, the State Board's priority chemical study of pentachlorophenol was expanded to include monitoring for CDDs and CDFs in dip tanks and other locations at sawmills and to investigate contaminant levels at wood treatment plants.

NOMENCLATURE OF CDDs AND CDFs

Although the term "dioxin" has become synonymous with 2,3,7,8-tetrachlorodibenzo-p-dioxin, "dioxin" is not used elsewhere in this document because the subject of this report is not one but a number of different CDDs. The nomenclature of CDDs and CDFs is important because there are enormous differences in toxicity between compounds. Those compounds chlorinated at the 2,3,7, and 8 positions and containing from four to seven chlorine atoms are believed to be most toxic. In this report, these CDDs and CDFs are referred to as 2,3,7,8 chlorine-substituted compounds or, more simply, as 2,3,7,8 congeners.

The basic skeleton of all the CDDs is dibenzo-p-dioxin, a molecule containing two benzene rings joined by two oxygen atoms (Figure 1.1). The dibenzo-p-dioxin molecule is chlorinated if a chlorine atom is attached to any of the positions numbered 1 through 4 and 6 through 9. The dibenzo-p-dioxin skeleton can accommodate up to eight chlorine atoms. 2,3,7,8-Tetrachlorodibenzo-p-dioxin contains four chlorine atoms, one each at the 2,3,7, and 8 positions (Figure 1.1). For purposes of simplification, 2,3,7,8-tetrachlorobdibenzo-p-dioxin is abbreviated as 2,3,7,8-tetraCDD in this document. Numbers indicate location of chlorine atoms on the molecule and tetra refers to four chlorines. Other four chlorine dibenzo-p-dioxins can also occur, for example 1,4,6,9-tetraCDD. In fact, there are 22 different ways that four chlorines can be arranged on the molecule; in chemical terminology there are 22 different "isomers".

There are three terms of chemical nomenclature that are used in this document to characterize CDDs: isomer group, isomer, and congener. CDDs can be divided into eight groups called isomer groups (also called homologues), with each isomer group containing the same number of chlorine atoms. For example, tetraCDD is the four chlorine isomer group of CDDs. An isomer is defined by the arrangement of chlorine atoms within an isomer

TABLE 2.1 (continued)

SUMMARY OF PHOTOTRANSFORMATIONS OF CDDs AND CDFs

	Process	Medium	Compound or Precursor	Fate or Product	Reference
B.	Solid Phase	wood, UV or sunlight	higher CDD	lower CDD (preferential peri chlorine removal	Lamparski et al., 1980
		wood, UV or sunlight	octaCDD	1,2,3,4, 6,7,8-heptaCDD (major product)	Lamparski et al., 1980
C.	Gamma irradiation	benzene-hexane, gamma irradiation	octaCDD octaCDF	lower CDDs and CDFs (non-preferential chlorine removal)	Buser, 1976

group. The CDDs 1,4,6,9-tetraCDD and 2,3,7,8-tetraCDD are isomers within the tetraCDD isomer group because both CDDs contain 4 chlorines. The term CDD congener refers to any CDD compound. For example, the CDDs 2,3,7,8-tetraCDD and 1,2,3,7,8-pentaCDD both are highly toxic. They are both CDD congeners but are not isomers because one contains four chlorine atoms and the other has five chlorines. There are a total of 75 CDD congeners (Table 1.1).

Similar nomenclature refers to the dibenzofurans. Because there is only one oxygen atom in the skeleton (Figure 1.1), the molecule is less symmetrical, and there are more congeners due to a larger number of possible chlorine arrangements. For example, while there are 22 tetraCDD isomers, there are 38 tetraCDF isomers. There are a total of 135 congeners in the eight CDF isomer groups.

In this report, as stated above, 2,3,7,8 chlorine-substituted compounds also are referred to as 2,3,7,8 congeners. While public health and environmental concerns in the United States initially focused on 2,3,7,8-tetraCDD, there has been increasing awareness that presence of the other 2,3,7,8 congeners among the CDDs and CDFs should also be closely monitored.

The CDDs and CDFs have not been deliberately manufactured except for use as laboratory standards to confirm chemical analyses. Rather, these compounds appear as by-products of chemical synthesis, electrical equipment fires, municipal incineration of solid wastes and other causes (See Appendix B "Sources"). Although 2,3,7,8-tetraCDD has not been reported in pentachlorophenol formulations manufactured in the United States, other 2,3,7,8 CDD and CDF congeners are present in commercial PCP formulations.

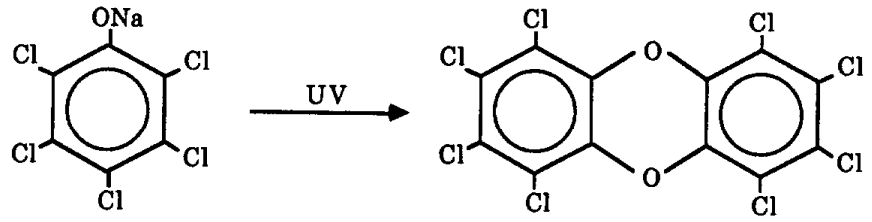
CALIFORNIA WATER RESOURCES CONTROL BOARD STUDY

By late 1984, the State Board had sampled for presence of CDDs and CDFs at several sawmills with emphasis on soils, dip tank liquids, and sludges. The results showed that CDDs and CDFs were present where pentachlorophenol had been used for wood preservation. The chemical analyses were for isomer groups (isomer group analysis); concentrations were reported as 50 ppb "hexaCDD", for example, without identification of specific isomers within an isomer group. The State Board informed other agencies of the findings, and an interagency group subsequently met with the Secretary of Environmental Affairs. The consensus of the meeting was that the findings were provocative and of concern, but confirmation of results was necessary. The interagency group agreed with State Board staff that, if possible, future studies should attempt to: (1) validate results

FIGURE 2.2

CDD AND CDF FORMATION REACTIONS

A. PCP to OctaCDD

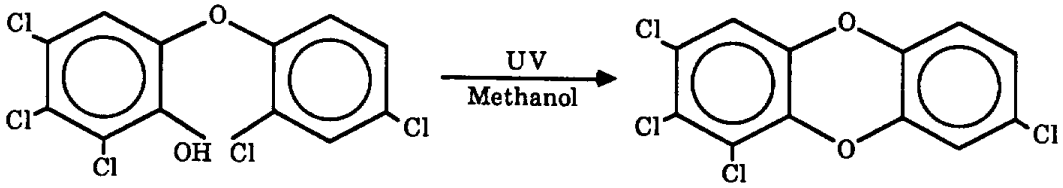


Sodium Pentachlorophenate

Octachlorodibenzo-p-dioxin

(Adapted from Crosby et al., 1973)

B. Prediоxin to CDD

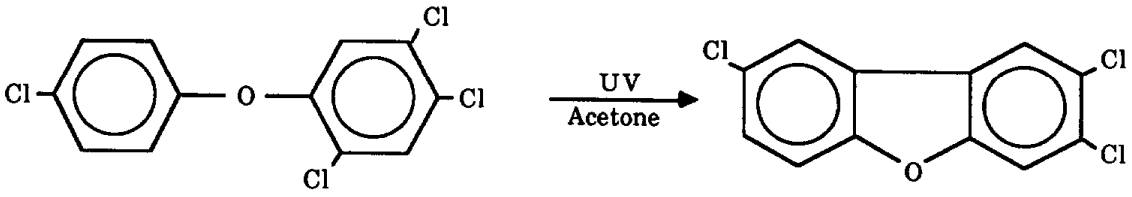


4,5,6-Trichloro-2-(2,4-dichlorophenoxy) phenol
"Prediоxin"

1,2,3,8-TetraCDD

(Adapted from Nilsson et al., 1974)

C. Chlorinated Diphenyl Ether to CDF



2,4,4',5-Tetrachlorodiphenyl Ether

2,3,8-TriCDF

(Adapted from Choudhry and Hutzinger, 1982)

FIGURE 2.3

PHOTOLYTIC DECHLORINATION
(From NRCC, 1981)

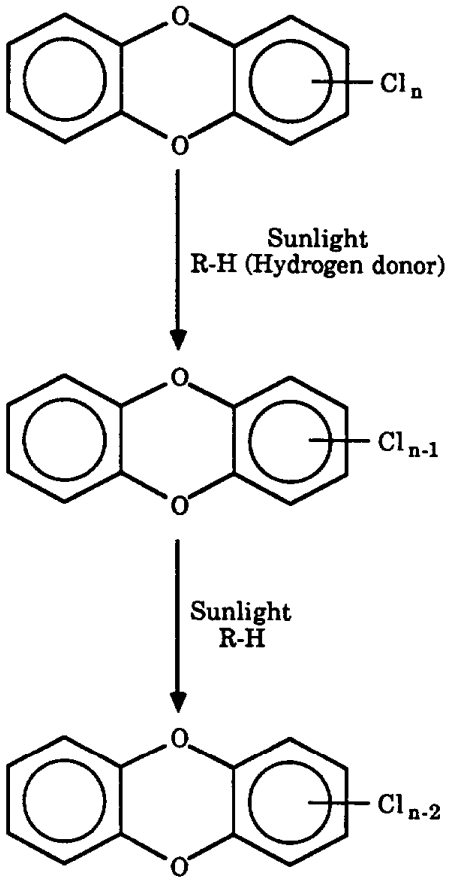
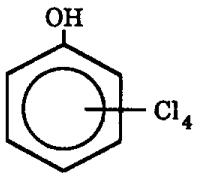
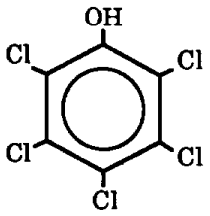


FIGURE 2.1

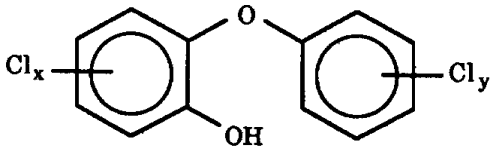
COMPOUNDS PRESENT IN COMMERCIAL FORMULATIONS OF CHLOROPHENOLS THAT MAY BE TRANSFORMED TO CDDS AND CDFS



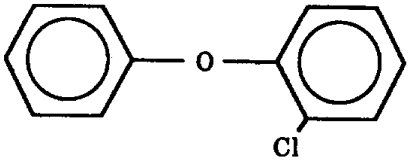
Tetrachlorophenol



Pentachlorophenol



"Predioxin"
Chloro-2-phenoxyphenol



Polychlorinated Diphenyl Ether (PCDE)
(2 - Chlorodiphenyl Ether)

FIGURE 2.4

PHOTOLYTIC DECHLORINATION OF OctaCDD (SOLUTION PHASE)
(Data from Buser and Rappe, 1978)

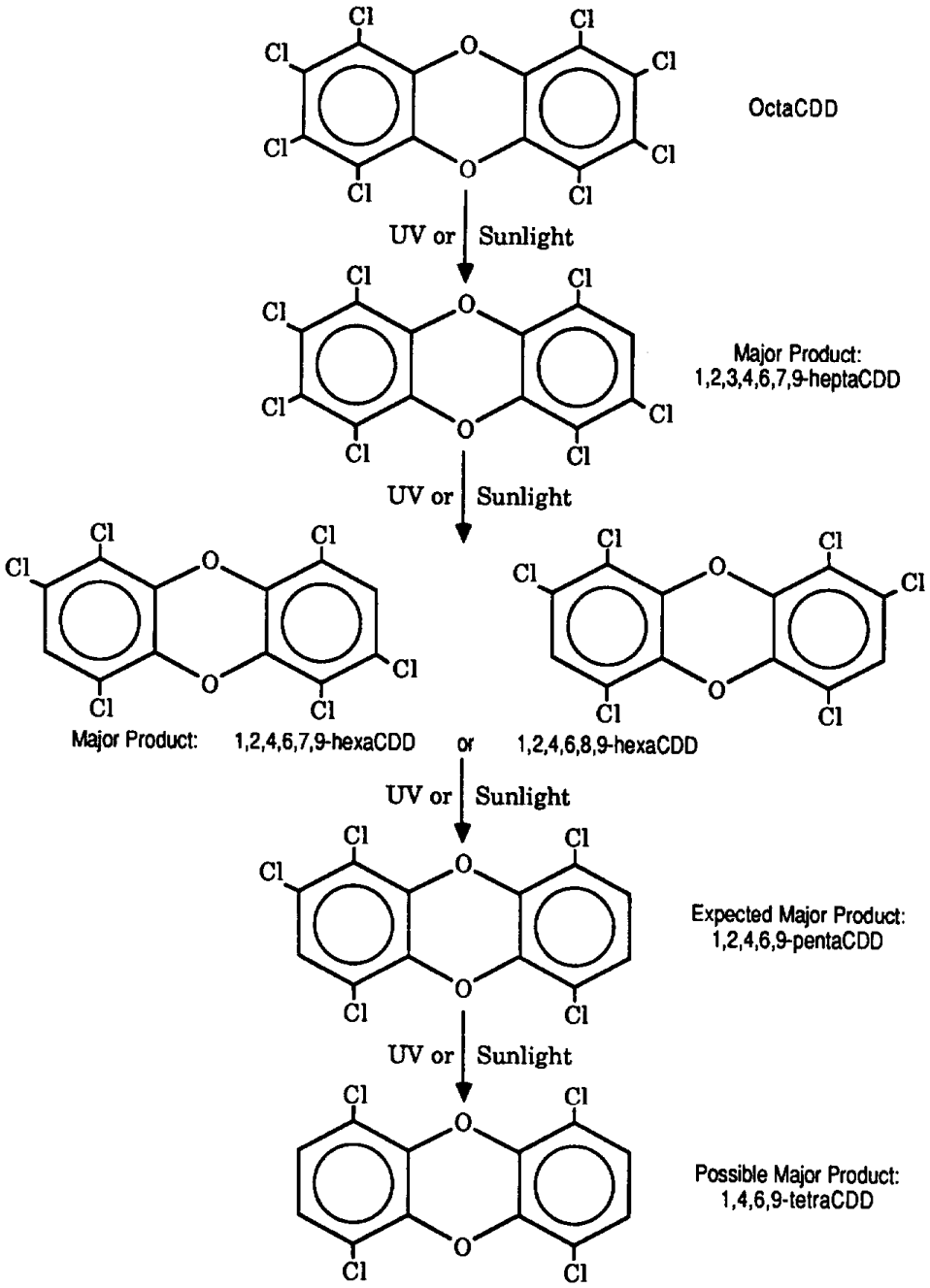


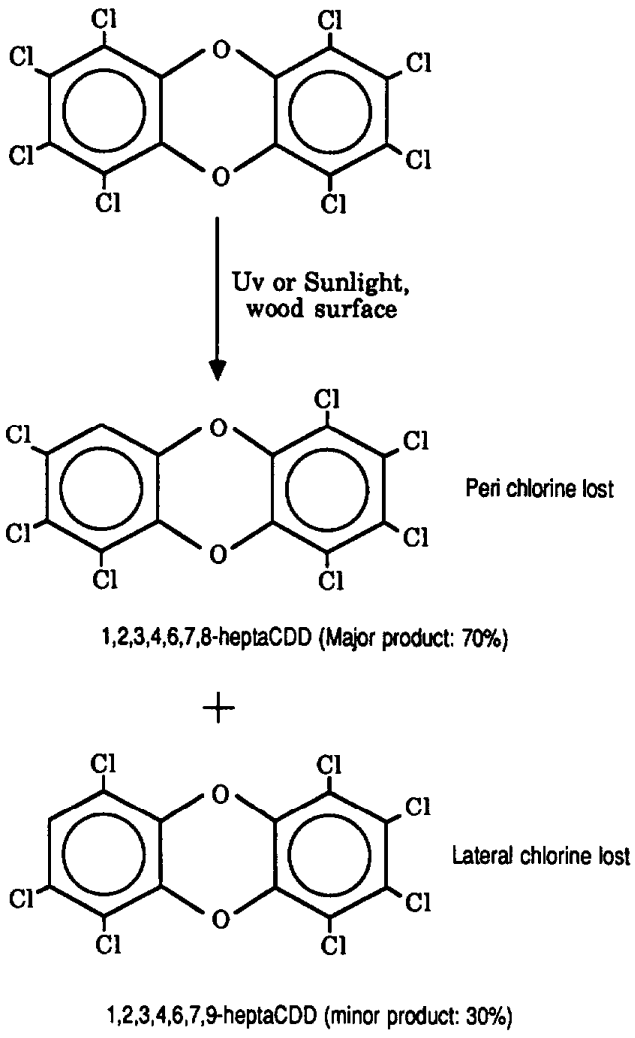
TABLE 2.1 (continued)

SUMMARY OF PHOTOTRANSFORMATIONS OF CDDs AND CDFs

Process	Medium	Compound or Precursor	Fate or Product	Reference
2.	benzene-hexane, UV	1,2,3,6, 7,8-hexaCDD	1,2,3,6,8-pentaCDD (major pentaCDD), 1,3,6,8-tetraCDD (major tetraCDD), 2,3,7,8-tetraCDD (trace)	Buser, 1979
3.	benzene-hexane, 310nm UV	1,2,3,7, 8,9-hexaCDD	1,2,3,7,9-pentaCDD (major pentaCDD), 1,3,7,9-tetraCDD (major tetraCDD), 2,3,7,8-tetraCDD (trace)	Buser, 1979
4.	methanol, 310nm UV	octaCDF	mixture of tetra to OctaCDF	Hutzinger et al., 1973
5.	hexane, 254nm UV	tetraCDFs	triCDFs	Mazer and Hileman, 1982
6.	methanol 310nm UV	2,8-diCDF	2-monoCDF	Crosby et al., 1973
7.	benzene-hexane, UV	octaCDF	heptaCDFs (all 4 isomers) hexaCDFs (13 of 16 possible isomers)	Buser, 1976

FIGURE 2.5

PHOTOLYTIC DECHLORINATION OF OctaCDD (SOLID PHASE)
(WOOD SURFACE - LOSS OF PERI CHLORINES)
(Data from Lamparski et al., 1980)



1,2,3,4,6,7,8-heptaCDD (Major product: 70%)

+

1,2,3,4,6,7,9-heptaCDD (minor product: 30%)

(2,3,7,8-substituted) 1,2,3,7,8-pentaCDD and no 2,3,7,8-tetraCDD (medium not described). Based on these and other studies, the U.S. EPA (1985b) concluded that it is unlikely that photolysis of octaCDD and heptaCDD will produce CDDs chlorinated at all four of the 2,3,7, and 8 positions on the dibenzo-p-dioxin molecule. In other words, breakdown of more highly chlorinated compounds would result in less toxic CDDs.

The U.S. EPA (1985b) conclusion failed to distinguish between laboratory studies and environmental conditions. The fact that irradiation was performed in the presence of hydrogen-donor solutions means that the data can not be directly extrapolated to other environments. Choudhry and Hutzinger (1982) have noted that the type of medium in which CDDs are exposed will affect which chlorines are removed by photolysis. In organic solutions, as noted above by Buser and Rappe (1978), the lateral (2,3,7, and 8) chlorines are removed preferentially. However, on solid media, such as wood surfaces, the peri position (1,4,6 and 9) chlorines are more likely to be removed from octaCDD. This difference was observed when Lamparski et al. (1980) irradiated pentachlorophenol-treated wood under conditions of natural and artificial sunlight. On wood treated with purified pentachlorophenol (very low levels of contaminants), octaCDD was formed by photolytic condensation of pentachlorophenol and subsequently dechlorinated to heptaCDDs and hexaCDDs. The dominant heptaCDD was the 1,2,3,4,6,7,8 isomer (Figure 2.5). Lamparski et al. (1980) stated that "OCDD present on a wood surface is somehow activated so that the preferential chlorine loss occurs at the peri position rather than the lateral position that Buser observed in solution." In this solid phase study, breakdown of higher CDDs resulted in more toxic compounds.

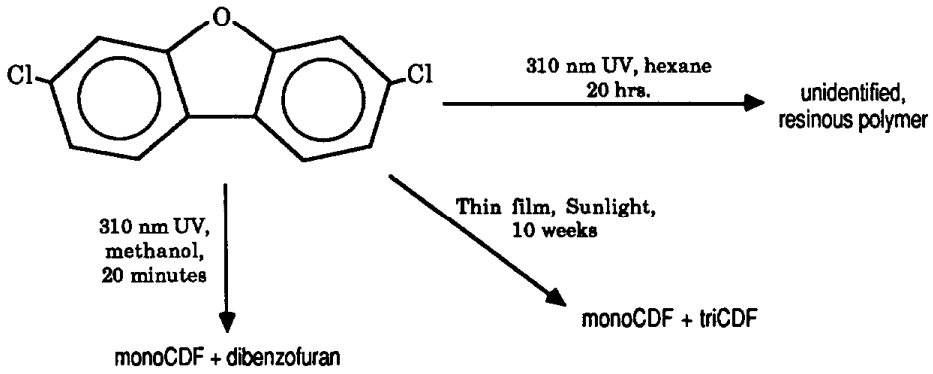
Nestrick et al. (1980) compared photolysis rates for tetraCDD isomers irradiated on a glass surface (solid phase) and in a dilute hydrocarbon solution (solution phase). They examined all 22 tetraCDD isomers and ranked the isomers by relative half-lives. The 2,3,7,8-tetraCDD (all lateral chlorines) had the shortest half-life in a hydrocarbon solution and the longest half-life when exposed as a thin film on a glass surface. In solution phase, the half-life of 2,3,7,8-tetraCDD was 57 minutes; the solid phase half-life was 8,400 minutes. In contrast, the 1,4,6,9 isomer (all peri position chlorines) had the longest half-life in solution. A similar phase-dependent reversal of half-lives was noted for some hexaCDDs and the two heptaCDDs. In hydrocarbon solution, the 1,2,3,6,7,8-hexaCDD had a 50 percent shorter half-life than a mixture of 1,2,3,6,7,9-/1,2,3,6,8,9-hexaCDD. On a glass surface, the half-life of 1,2,3,6,7,8-hexaCDD was more than five times longer than the mixture. Of the two heptaCDDs, 1,2,3,4,6,7,8-heptaCDD had a shorter half-life in

FIGURE 2.6

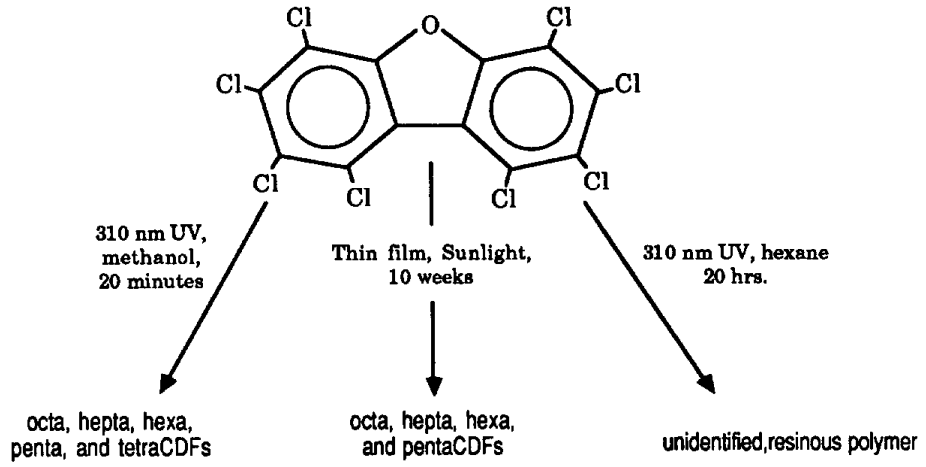
PHOTOLYTIC TRANSFORMATIONS OF PCDFs

(adapted from Huntzinger et al., 1973)

(i) Photolysis of 2,8-diCDF



(ii) Photolysis of octaCDF



solution and a longer half-life on glass than did 1,2,3,4,6,7,9-heptaCDD. It is clear that more studies are necessary to determine specific congeners formed by photolysis of higher CDDs, studies performed both in solution phase and on solid phases such as wood and soil.

Buser (1976) irradiated octaCDD dissolved in a benzene-hexane solution with both gamma rays and UV and compared the dechlorination products. Whereas UV photolysis resulted in preferential loss of lateral chlorines (formation of 1,2,3,4,6,7,9-heptaCDD), gamma irradiation was non-specific and resulted in about equal formation of 1,2,3,4,6,7,8- and 1,2,3,4,6,7,9-heptaCDDs.

CDFs

Less information is available on CDF breakdown reactions. Buser (1976) irradiated octaCDF dissolved in a solution of benzene and n-hexane with both UV and gamma radiation and noted that dechlorination to lower CDFs seemed to be the major reaction pathway. Crosby et al. (1973) reported that 2,8-diCDF dissolved in methanol was rapidly photodegraded to 2-monoCDF under laboratory conditions. Hutzinger et al. (1973) reported the rapid UV photolysis of both 2,8-diCDF and octaCDF in methanol and hexane solutions, resulting in dechlorination with eventual accumulation of "unidentified resinous polymeric-products". The authors noted that the similar photolytic rates in hydrogen-donating solvents for diCDF and octaCDF contrasts with the CDD work of Crosby et al. (1971) where 2,7-diCDD was photodegraded much more rapidly than octaCDD in methanol. In contrast to rapid photolytic rates in hydrogen-donating solvents (20 minutes), transformation rates of thin films of 2,8-diCDF and octaCDF exposed to sunlight in the presence of water were much slower, two months (Figure 2.6; Hutzinger et al., 1973).

Mazer and Hileman (1982) examined the photolysis by UV radiation of 254 nm wavelength in hexane or tetradecane of eight tetraCDFs and derived three guidelines for predicting chlorine loss as tetraCDFs are converted to triCDFS:

1. Chlorines will be removed from the most highly chlorinated ring.

2. The greater the number of adjacent chlorines to a chlorine atom, the greater the likelihood of losing that chlorine.

3. Given an equal number of chlorines at the 2 and 3 positions, the 3 position chlorine will be lost first.

VOLATILIZATION

Very little information is available on the importance of volatilization for CDF environmental fate, due to lack of water solubility and vapor pressure data (NRCC, 1984). The CARB and CDHS (1986) noted that, as the number of chlorine atoms increases in CDDs and CDFs, the vapor pressures and water solubilities decrease. Thus, the more highly chlorinated the CDD or CDF, the lower expected rate of volatilization (U.S. EPA, 1985a).

The U.S. EPA (1985a) summarized several studies performed on microbial systems and noted that loss by volatilization was considered a major factor in 2,3,7,8-tetraCDD loss. However, there were no quantitative data on rate of volatilization provided in the studies.

Corbet et al. (1983) reported that the highest volatilization rate of radiolabelled 1,3,6,8-tetraCDD from an outdoor pond occurred in the first 24 hours. Marcheterre et al. (1985) reported similar findings for octaCDD.

In a recent review, Mill (1985) stated that volatilization of 2,3,7,8-tetraCDD from soil will be very slow over a wide range of soil moisture content because of low vapor pressure and water solubility. In a study of an herbicide production facility, Thibodeaux (1983) suggested that 29 percent to 46 percent of 2,3,7,8-tetraCDD volatilized from soil over a 350 day period. Muir et al. (1985) studied the fate of radiolabelled 1,3,6,8-tetraCDD in field plots of soil and found that reduction in radioactivity was not due to movement or degradation. They suggested that losses may have occurred by volatilization.

Computer models discussed below have estimated that vaporization will be the major source of loss of 2,3,7,8-tetraCDD from both aqueous and soil systems. In a model predicting 2,3,7,8-tetraCDD loss from aqueous systems, the NRCC (1981) attributed 100 percent of the loss to volatilization, with no appreciable losses to biodegradation or photolysis.

Freeman and Schroy (1985) developed a model to predict the vapor phase migration in soil columns of chemicals possessing low vapor pressures. They applied the model to soil contaminated by 2,3,7,8-tetraCDD in Times Beach, Missouri and made several conclusions about the behavior of this CDD congener:

1. The compound is volatile and its migration through and out of soil is temperature dependent.

The National Research Council of Canada (1984) discussed the Mazer and Hileman study and noted that, since 254 nm ultraviolet radiation does not reach the earth's surface, the guidelines have "little bearing" on environmental conditions.

MICROBIAL DEGRADATION

Published information on microbial degradation of CDDs is limited (NRCC, 1984). The U.S. EPA (1985a) cited reports that demonstrated lack of microbial degradation of 2,3,7,8-tetraCDD in aquatic systems and by inference concluded that the penta- and hexaCDDs would be even more resistant to microbial breakdown. The biotransformation and biodegradation half-life of 2,3,7,8-tetraCDD in aquatic systems is greater than one year (U.S. EPA 1979a). Some investigators have reported limited microbially mediated degradation of 2,3,7,8-tetraCDD.

Huetter and Philippi (1982) reported very slow microbial degradation of 2,3,7,8-tetraCDD in soil and liquid systems. They found that approximately 1 percent of radiolabelled 2,3,7,8-tetraCDD was converted by single and mixed microbial cultures to an unidentified polar metabolite after several months of incubation. Matsumura and Benezet (1973) examined the ability to degrade 2,3,7,8-tetraCDD of 100 microbial strains known to breakdown resistant pesticides and found only five strains performed any degradation. Quensen and Matsumura (1983) reported oxidative metabolism of 2,3,7,8-tetraCDD present at 5 ppb by two bacterial species and found that metabolism was stimulated by use of ethyl acetate as a carrier solvent. The authors suggested that the solvent may exert a stimulatory effect by aiding 2,3,7,8-tetraCDD penetration of the cell membrane. Thus, restricted microbial uptake could be a limiting factor for environmental degradation of this compound.

Bumpus et al. (1985) recently reported that a common white-rot fungus (Pharnerochaete chrysosporium) is capable of oxidizing 2,3,7,8-tetraCDD to carbon dioxide and attributed this ability to secretion of an extracellular lignin-degrading enzyme system when the fungus was grown in nitrogen-, carbohydrate-, or sulfur-deficient cultures. By showing that this fungus could also convert several other compounds such as DDT and polychlorinated biphenyls to carbon dioxide, the authors demonstrated that this organism is capable of cleaving the ring structure of several halogenated aromatic hydrocarbons. They concluded that, because the degradation is initiated by nitrogen deficiency rather than the level of 2,3,7,8-tetraCDD present, low levels of environmental 2,3,7,8-tetraCDD may provide sufficient substrate for biodegradation.

The U.S. EPA (1985a) estimated a soil to water partition coefficient of 48,000 to 1 for soil containing ten percent organic matter. Due to this high affinity for soils, the 2,3,7,8-tetraCDD congener is expected to remain bound at or near the soil surface and apparently becomes more difficult to desorb over time (U.S. EPA 1985a). DiDomenico et al. (1980b) examined 2,3,7,8-tetraCDD levels at 44 contaminated sites at Seveso, Italy: one month after the Seveso chemical plant accident, the 2,3,7,8-tetraCDD half-life in soil was estimated to be on the order of one year. Seventeen months after the incident, the authors estimated the half-life to be greater than ten years. In the previous discussion on volatilization, Freeman and Schroy (1985) concluded that the persistence in soil of 2,3,7,8-tetraCDD could not be expressed as a simple half-life. DiDomenico et al. (1980a) also measured the amount of downward migration at Seveso sites and found 2,3,7,8-tetraCDD at depths of up to 30 cm. However, the highest levels were detected in soil from 0.5 cm to 1.5 cm below the surface.

Freeman and Schroy (1986) modeled the movement of 2,3,7,8-tetraCDD at a site where an herbicide (Agent Orange) contaminated with this compound had been buried. They concluded that vertical migration was very slow, only 10 centimeters over a period of 12 years. Kitunen et al. (1987) examined soil and contamination by chlorinated phenols at four sawmills and reported that while chlorinated phenols were mobile and migrated downward, the CDF (0.2 to 5 ppm) and polychlorinated phenoxyphenol (1 to 50 ppm) contaminants were contained in the top layer of soil.

Although several reviews and models, some cited above, have commented that CDDs are relatively immobile in soils and unlikely to migrate for appreciable distances, recent empirical studies have noted ground water contamination by CDDs and CDFs. Pereira et al. (1985) reported ground water and porous media contaminated by CDDs at depths up to 30 meters at a site in Florida. CDDs and CDFs in both a confined and unconfined aquifer, as well as in soil cores from depths of up to 16 meters, have been detected at Visalia, California (see Chapter 5 of this report).

Recent studies have begun to elucidate these empirical observations of CDD and CDF migration to ground water. Nkedi-Kizza et al. (1985) have noted that at most waste disposal and land treatment sites, soil solutions will consist of both water and mixtures of organic solvents. Most data currently used to predict migration are for sorption of hydrophobic organic compounds from aqueous solution rather than from water-organic solvent mixtures. For sites where organic solvents are present, soil sorption should be characterized by mixtures of water and organic solvent (Nkedi-Kizza et al., 1985).

2. Because the migration of 2,3,7,8-tetraCDD is highly dependent on depth in the soil column, the environmental fate can not be represented as a simple half-life. For example, the apparent half-life on a soil surface would be on the order of weeks; in contrast, the half-life below a depth of 5 cm would be expressed as years.

3. The compound will volatilize rapidly during summer months, but volatilization will be negligible during the winter.

4. During the first summer after 2,3,7,8-tetraCDD was applied to Times Beach soil, 90 percent of the upper 1 cm layer and 50 percent of the total amount volatilized.

The fate of CDDs and CDFs in the atmosphere is largely unknown because there have been no studies reported on either photochemistry or atmospheric chemistry (CARB and CDHS, 1986). The CARB (1986) noted that these compounds are emitted to the atmosphere in two forms: in the vapor phase and adsorbed to particulates. The fate and persistence will depend in part on the compounds' physical state; the California Air Resources Board (CARB and CDHS, 1986) concluded that CDDs and CDFs will tend to adsorb to particulate matter because of their low vapor pressure.

PERSISTENCE AND MOVEMENT IN SOIL AND SEDIMENTS

CDDs and CDFs are believed to sorb strongly to soils and sediments (U.S. EPA 1985a), and most models predict that soils and sediments will serve as the major sink for these compounds.

Soils

In the absence of organic solvents, leaching and downward migration of CDDs and CDFs to ground water is unlikely (Hutzinger et al., 1985). However, in the presence of organic solvents or in areas possessing sandy soils of low organic content, a greater degree of vertical migration may occur.

CDDs and CDFs in contaminated soil can be spread laterally by wind and soil erosion (Hutzinger et al., 1985). Due to the estimated high affinity of CDFs for soil systems, the NRCC (1984) predicted that these compounds will be highly persistent in the environment. Young (1981) estimated that only about one percent of the 2,3,7,8-tetraCDD remained in an aerial test application site 14 years after spraying. However, Young (1981) noted that, once this compound has bound to soil, its persistence becomes enhanced significantly.

PLANT UPTAKE

Although removal of CDDs and CDFs from contaminated soils by plant uptake has been proposed as a soil clean-up technique (see Pesticide and Toxic Chemical News, Sept. 18, 1985, p. 12), the data on uptake by terrestial plants are equivocal. The U.S. EPA (1985a) concluded that 2,3,7,8-tetraCDD present in contaminated soil is "not likely" to concentrate in terrestial plants. In contrast to soil systems, the agency (U.S. EPA, 1984a; U.S. EPA, 1985a) cites studies reporting bioaccumulation of this congener by aquatic plants. The National Research Council of Canada's extensive reviews on CDDs (NRCC, 1981) and CDFs (NRCC, 1984) do not address plant uptake. A description of representative terrestrial and aquatic plant studies is provided below.

Terrestial Plants

Several studies have examined vegetable and fruit crop uptake following the accidental 2,3,7,8-tetraCDD release at Seveso, Italy. The Seveso studies have provided contrasting observations. An early study by Coccuci et al. (1979) concluded that plants take up this congener and translocate it to leaves and fruits. In contrast, Wipf et al. (1982) noted that trace levels of 2,3,7,8-tetraCDD were present only on outer surfaces of Seveso fruits and vegetables and attributed the source of this CDD to contaminated dust rather than plant uptake. Recently, in a study using a mixture of contaminated and uncontaminated Seveso soil, Facchetti et al. (1985) reported uptake by vegetables in their root systems. For example, the roots of corn grown in soil with levels at 0.75 ppb 2,3,7,8-tetraCDD contained 1.0 ppb after the roots were rinsed in hexane. However, the authors found only a few ppt in upper portions of vegetables, and they concluded that these low levels of 2,3,7,8-tetraCDD present in above ground parts were transported by volatilization from the soil rather than translocation within the plant.

Young (1981) examined a field sprayed with Agent Orange, an herbicide that contained 2,3,7,8-tetraCDD as a contaminant, and measured 2,3,7,8-tetraCDD levels in roots, stems, and leaves. Young (1981) reported levels in roots and soil were similar at approximately 750 ppt. Levels of 2,3,7,8-tetraCDD in leaves ranged from one to ten percent of root levels. Young did not determine if leaf levels resulted from plant uptake or from contamination by soil particles.

Jackson et al. (1985) examined the leaching potential of 2,3,7,8-tetraCDD in soils contaminated with chlorinated semi-volatile organic compounds and compared leaching to that in "clean" soils that had been spiked with 2,3,7,8-tetraCDD. There was a strong correlation of leaching potential with solvent-extractable organic content in the soils. They suggested that the presence of halogenated semivolatile compounds as co-contaminants has a major role in regulating 2,3,7,8-tetraCDD solubility and migration in contaminated soil.

Enfield (1985) noted that relatively immobile compounds have been observed to migrate faster than predicted by hydrophic theory. If two percent of total soil fluid is an organic fraction, then hydrophobic theory may underestimate soil mobility of these compounds by a factor of greater than 100. According to Enfield (1985), a partitioning of organic chemicals occurs between water and dissolved organic material. At certain waste disposal sites, both an organic fluid phase and an aqueous phase have been observed to flow through the soil, with the organic phase aiding the transport of hydrophobic chemicals. In his model, Enfield (1985) noted that increased mobility of CDDs is predicted at levels of organic carbon found in the environment (5 to 10 mg/l).

Sediments

Laboratory experiments indicate that 2,3,7,8-tetraCDD is highly sorbed to biological and sediment matrices and more highly chlorinated CDDs are predicted to be concentrated in sediments (U.S. EPA, 1985a). In computer simulated models of an oligotrophic lake and eutrophic pond, the NRCC (1981) determined that the major sinks for 2,3,7,8-tetraCDD would be suspended particles and sediments and that the larger the mass of these sinks, the longer this congener would persist in the environment (NRCC, 1984).

Karickhoff and Morris (1985) discussed the kinetics of sorption of hydrophobic chemicals in sediments and noted that sorption phenomena are frequently described as rapidly reaching equilibrium and as readily reversible. However, sorption under field conditions of highly hydrophobic compounds frequently requires days to weeks in order to reach equilibrium. Karickhoff and Morris (1986) propose a two compartment model, one rapid and readily reversible compartment and a second, slow to reach equilibrium. This approach may be useful to describe the behavior of CDDs and CDFs in sediments.

TABLE 2.2

SUMMARY OF ENVIRONMENTAL FATE OF CDDs AND CDFs

Process	Medium	Compound or Precursor	Fate or Product	Reference
1. Phototransformation				
a. Formation				
	water,UV	Na-PCP	octaCDD	Crosby et al., 1973
	wood, sunlight	PCP	octaCDD	Lamparski et al., 1980
	methanol, UV	predioxins	CDDs	Nilsson et al., 1974
	hexane, UV	chlorinated diphenyl ethers	CDFs	Norstrom et al., 1976 and 1977
b. Photolysis				
	solution phase: organic solvents, UV or sunlight	higher CDDs and CDFs	lower CDDs and CDFs: preferential removal of lateral (2,3,7,8) chlorines	Buser and Rappe, 1978
	solid phase: wood and glass surfaces, UV or sunlight	higher CDDs	lower CDDs preferential removal of peri (1,4,6,9) chlorines	Lamparski et al., 1980
	benzene-hexane, gamma irradiation	octaCDD octaCDF	lower CDDs and CDFs: non-preferential removal of chlorines	Buser, 1976

Aquatic Plants

Plant uptake of CDDs in aquatic systems appears to be greater than in terrestial systems. Further, studies are more in agreement for aquatic systems. Tsushimoto et al. (1982) reported rapid accumulation of radioactive 2,3,7,8-tetraCDD by pondweeds. Outdoor ponds were dosed with 54 ppt 2,3,7,8-tetraCDD, and a maximum of 7,000 ppt was concentrated in pondweeds after five days. An equilibrium level of 2,500 ppt in pondweeds was reached after a month. Corbet et al. (1983) reported concentration of radioactive 1,3,6,8-tetraCDD by both floating duckweed and rooted aquatic plants. Maximum levels of the 1,3,6,8-tetra isomer in rooted plants were reached at eight days. Yockim et al. (1978) reported a maximum bioconcentration factor of 2,083 at seven days for 2,3,7,8-tetraCDD by a freshwater alga.

SUMMARY AND DISCUSSION

A summary of environmental fate information for CDDs and CDFs is provided in Table 2.2.

Phototransformation

The CDDs and CDFs can be both formed and broken down by either artificial UV light or sunlight containing UV wavelengths. Under proper conditions, octaCDD can be formed from pentachlorophenol and subsequently dechlorinated to lower chlorinated CDDs. Certain contaminants present in commercial chlorinated phenols can be converted to CDDs and CDFs: predioxins to CDDs and polychlorinated diphenyl ethers to CDFs.

When exposed to ultraviolet (UV) light, CDDs and CDFs will undergo photolysis at a significant rate in the presence of an organic, hydrogen-donating substrate (Crosby et al., 1971). The presence of a hydrogen-donor, which in a number of laboratory experiments has consisted of methanol or hexane, is necessary for a significant amount of photolysis to occur. These conditions may not be met in most environmental situations. The reaction is slow in water and does not occur either on thin layers of pure tetraCDD or on dry soil surfaces (Crosby et al., 1973). The more highly chlorinated CDDs are less reactive than those which are less chlorinated (NRCC, 1981). It has been hypothesized that the low solubility of the more chlorinated CDDs and CDFs in water may retard their photolysis, whereas in an organic hydrogen-donating solvent, the necessary conditions for rapid photolysis are present (Crosby et al., 1981).

TABLE 2.2 (continued)

SUMMARY OF ENVIRONMENTAL FATE OF CDDs AND CDFs

Process	Medium	Compound or Precursor	Fate or Product	Reference
5. Plant Uptake				
a. terrestrial plants	soil	2,3,7,8-tetraCDD	uptake by roots	Facchetti et al., 1985
b. aquatic plants and algae	water	2,3,7,8-tetraCDD	uptake with maximum concentration at 5 days	Tsushimoto et al., 1982
		1,2,6,8-tetraCDD	uptake with maximum concentration at 8 days	Corbet et al., 1983

TABLE 2.2 (continued)

SUMMARY OF ENVIRONMENTAL FATE OF CDDs AND CDFs

Process	Medium	Compound or Precursor	Fate or Product	Reference
2. Microbial Degradation				
a. fungal catabolism (white rot fungus)	culture medium	2,3,7,8-tetraCDD	carbon dioxide	Bumpus et al., 1985
b. bacterial catabolism	culture medium	2,3,7,8-tetraCDD	unidentified polar metabolite(s)	Huetter and Philippi, 1982; Quensen and Matsumura, 1983
3. Volatilization				
computer simulation	aquatic pond and lake	2,3,7,8-tetraCDD	2,3,7,8-tetraCDD in vapor phase (atmosphere)	NRCC, 1981
4. Persistence and Movement				
a. computer simulation	aquatic pond and lake	2,3,7,8-tetraCDD	bound to sediment and suspended particles	NRCC, 1981
b. migration	waste site containing organic solvents	2,3,7,8-tetraCDD	will migrate with organic fraction, more mobile than predicted in spiked clean soils	Jackson et al., 1985

matter in the air. Summarizing other work, the CARB and CDHS (1986) stated that CDDs and CDFs appear stable when adsorbed to particulate matter, can migrate over great distances in the air, and are probably highly persistent in the atmosphere. Czucwa et al. (1984) found CDDs and CDFs in sediments from a lake located on Isle Royale in Lake Superior and concluded that their presence could only be explained by atmospheric deposition. Thus, while volatilization may remove CDDs and CDFs from aquatic and terrestial compartments, these compounds may be atmospherically transported and subsequently redeposited.

Persistence and Movement in Soil and Sediments

CDDs and CDFs are believed to adsorb strongly to soils, sediments, and biota. Sediments and suspended particulates will serve as sinks for these compounds in aquatic systems (NRCC, 1981); because of strong sorption, they will be highly persistent in the environment. As demonstrated at a site in Visalia, California, these compounds can travel considerable distances downward in soil if organic solvents are present (see Chapter 6: Monitoring). In the absence of organic solvents, CDDs and CDFs are not expected to migrate downward to any great extent. Recent evidence suggests that measurement of CDD movement in soil, based on spiking clean soil with CDDs, does not accurately portray migration at waste disposal and land treatment sites (Nkedi-Kizza et al., 1985; Enfield, 1985; Jackson et al., 1985).

Because these compounds will bind very tightly to organic material in soils and contaminated materials, CDDs and CDFs may escape detection in standard water leachate tests. Use of aqueous leachate tests for these compounds as a screening device prior to land disposal is inappropriate. Rather, a leachate mixture composed of water and organic solvents should be developed and used to determine levels of CDDs and CDFs (Nkedi-Kizza et al., 1985; Jackson et al., 1985). Thus, use of standard leachates required under RCRA for dioxin-containing wastes (U.S. EPA, 1986b) is probably inappropriate because it will underestimate concentrations of CDDs and CDFs in contaminated soils. The treatment standard requires that waste found to contain any tetra-, penta-, or hexaCDD or CDF at levels of 1 ppb or higher in a standard leachate test be treated before land disposal. Young (1981) noted that when soil has been contaminated for several years, the extraction of 2,3,7,8-tetraCDD and subsequent chemical analysis is difficult. The aqueous leachates referenced in the RCRA regulations may not desorb CDDs and CDFs that are highly adsorbed to organic material. In order to extract CDDs and CDFs from soils for chemical analysis, organic solvents are required (see discussion

The National Research Council of Canada (NRCC, 1981) has commented that there is little quantitative evidence on rates of CDD photolysis in the natural environment; many studies have been limited to laboratory conditions, using UV wavelengths (less than 290 nanometers) that are screened out by the earth's atmosphere. The U.S. EPA (1985b) stated that photolytic breakdown of CDDs "is not likely to be of environmental importance" in water because these compounds are unlikely to receive UV radiation due to low UV penetration of surface waters and because sorption of CDDs on sediments and suspended particles effectively removes them from solution.

Microbial Degradation

Information on microbial degradation of CDDs and CDFs is very limited (NRCC, 1981; NRCC, 1984). Resistance to microbial attack will increase with increasing chlorination. Overall data support the view that CDDs and CDFs are highly resistant to microbial transformation. A recent report has indicated that a common fungus can break down 2,3,7,8-tetraCDD (Bumpus et al., 1985) under conditions of "nitrogen starvation". The potential role of this fungus in land treatment systems should be examined.

Volatilization

Despite the low vapor pressure of CDDs and CDFs, volatilization is now considered a potential source for loss of these compounds from environmental compartments. In simulating the fate of 2,3,7,8-tetraCDD in two model aquatic ecosystems, the National Research Council of Canada (NRCC, 1981) assigned 100 percent of loss to volatilization and zero to photolysis and microbial degradation. The U.S. EPA (1985b) noted that volatilization is a likely fate for CDDs in aquatic environments. Earlier studies had concluded that volatilization was not an important fate of 2,3,7,8-tetraCDD; for example, in the Water-Related Environmental Fate of 129 Pollutants, the U.S. EPA 1979) stated that volatilization is "probably not an important process" in the aquatic fate of this congener. For CDF volatilization, the NRCC (1984) noted an almost total lack of physical constant data but, by inference from CDD data, predicted low rate constants for volatilization. Nonetheless, the NRCC (1984) concluded that volatilization could play a role in environmental distribution.

Conversion of CDDs and CDFs to the vapor phase does not explain the ultimate fate of these compounds. The California Air Resources Board (CARB and CDHS, 1986) noted that there have been no studies on the behavior of CDDs and CDFs in the vapor phase and there is little knowledge of their fate in the atmosphere, but predicted these compounds would be sorbed to particulate

1. The fate of chlorinated phenols, predioxins, and polychlorinated diphenyl ethers should be examined during land treatment. This is because CDDs and CDFs can be formed from precursor compounds by sunlight under certain conditions. For example, Norstrom et al. (1976) noted the potential in the environment for CDF formation from polychlorinated diphenyl ethers, compounds which are present at up to 100 ppm in commercial pentachlorophenol.

2. Similarly, the dechlorination products from breakdown of higher CDDs and CDFs should be measured. While the U.S. EPA (1985b) noted that the potential for photoformation of 2,3,7,8-tetraCDD from higher chlorinated compounds was unlikely due to preferential removal of the lateral (2,3,7, and 8) chlorine atoms, the U.S. EPA comments appear to be based on a review of solution phase photolysis. In contrast, the findings of Lamparski et al. (1980) indicate that in solid phase photolysis, the potential exists for formation of more toxic CDDs and CDFs by removal of the peri (1,4,6, and 9) chlorine atoms. In land treatment, it should be determined if photodegradation reactions occur as solution phase, solid phase, or a mixture of phases.

3. If a reduction occurs in concentrations of CDDs and CDFs, the ultimate fate of these compounds requires investigation. While Dobbs and Grant (1979) predicted that the most susceptible CDD to photolysis would be 2,3,7,8-tetraCDD, Crosby et al. (1973) reported no 2,3,7,8-tetraCDD degradation in dry soil. Plimmer (1978) suggested that there would be little or no loss of 2,3,7,8-tetraCDD in dry soil from photolysis and that any loss occurring may come from volatilization. Migration by wind blown dust and soil may also account for CDD and CDF removal (Thibodeaux, 1983; Hutzinger et al., 1985).

4. The kinetics of CDD and CDF photolysis during land treatment requires study. Nestrick et al. (1980) noted that some 2,3,7,8-chlorinated CDDs have longer half-lives in solid phase than in organic solution. Solid phase half-lives of 2,3,7,8-tetraCDD and 1,2,3,6,7,8-hexaCDD were over 100 times longer than in organic solution phase. Further, these 2,3,7,8-chlorinated compounds were more resistant to solid phase photolysis than other tetra- and hexaCDDs irradiated.

5. Radiolabelled CDDs and CDFs should be employed during the land treatment study to determine a mass balance for environmental fate of these compounds. That is, the proportion

of chemical analysis in Appendix E). Similar extraction compounds should be used in leachate tests for detection of CDDs and CDFs.

Plant Uptake

There is consensus that plants in aqueous systems take up and concentrate CDDs and CDFs (U.S. EPA 1984a; U.S. EPA 1985b), although Kenaga and Norris (1983) have noted that these compounds may be adsorbed onto external surfaces of aquatic plants rather than actually taken up into plant cells. However, the data for uptake by terrestial plants are less clear. Studies of 2,3,7,8-tetraCDD concentrations in crops grown near the site of the Seveso, Italy chemical accident are contradictory: the presence of this congener in plants is variously attributed to plant uptake and translocation, contaminated dust, and volatilization from soil. After reviewing the literature, the U.S. EPA (1985b) concluded that 2,3,7,8-tetraCDD is "not likely to concentrate in plants grown on contaminated soils."

It should be noted that concentrations of 2,3,7,8-tetraCDD in contaminated soils where plant uptake was examined were on the order of one ppb. Levels of 2,3,7,8-chlorinated CDDs and CDFs encountered at California wood treatment plants and sawmills were much greater (in the high ppb to low ppm range as described in Chapter 5). An important research project would be to determine kinetics of plant uptake where high levels of CDDs and CDFs are present in soils. As Young (1981) has observed, animals foraging on CDD contaminated plants can potentially relocate these compounds off-site.

Land Treatment

In-situ land treatment has been proposed as a potential cleanup method for sites contaminated by CDDs, CDFs, and chlorinated phenols. This option has the potential advantage of being a relatively inexpensive method to clean large volumes of contaminated soil when compared to costs of thermal destruction or removal to distant landfills. Although the inherent assumption is that land treatment will be accomplished by photolysis and perhaps microbial degradation, Young (1981) has observed that reductions in CDD and CDF levels may involve off-site transport, including wind and water movement of contaminated particles, volatilization, and biomass removal. What is needed is a careful study of land treatment, which will examine a number of uncertainties including those listed below.

CHAPTER 3: AQUATIC TOXICOLOGY

While there are considerable data on the toxicity of some CDDs and CDFs to mammals, aquatic toxicity studies are few and mostly pertain to 2,3,7,8-tetraCDD toxicity.

The first section of this chapter addresses bioconcentration, metabolism and elimination of 2,3,7,8-tetraCDD and other CDDs and CDFs. The second discusses toxicity with the focus mainly on 2,3,7,8-tetraCDD due to lack of information on other CDDs and CDFs.

BIOCONCENTRATION

An organism's uptake and bioconcentration of toxic chemicals depends on factors such as the organism's food intake, surface area to weight ratio, characteristics of the medium in which the organism lives, molecular stability of the chemical, and the organism's metabolism and lipid content (Kenaga and Norris, 1983). The bioconcentration factor (BCF) is a constant proportionality that relates a specific chemical residue in an aquatic organism to the concentration of that chemical in water under standard conditions (Veith et al., 1980). BCFs can be a valuable means of estimating concentrations in water that would pose a threat to aquatic organisms and their consumers. To achieve this, the chemical concentrations in water and tissue must be accurately measured and toxic threshold concentrations in aquatic organisms and their consumers must be known.

Bioconcentration in Fish and Invertebrates

Based on the use of one measured and four estimated octanol/water partition coefficients (K_{ow}), EPA (1984a) predicted BCF values for 2,3,7,8-tetraCDD using six different equations that had been developed by Kenaga and Goring (1980); Veith et al. (1980); and Veith and Kosian (1983). The predicted BCF values derived from the measured partition coefficient (log K_{ow} = 6.15) ranged from 2,870 to 67,800. For the four estimated coefficients (log K_{ow} of 6.84 to 7.28), the predicted BCF values ranged from 6780 to 915,000. However, actual measured BCFs tend to fall at the low end of these predicted values (Table 3.1). The highest measured BCF of 2,3,7,8-tetraCDD reported in the literature for invertebrates is 9,222 in mosquito larvae, Aedes aegypti (Matsumura, 1977). It should be noted that in this study the concentration in water slightly exceeded the water solubility of 2,3,7,8-tetraCDD. The highest average BCF for 2,3,7,8-tetraCDD in fish was 9,270 for rainbow trout (Branson et al., 1985).

of these compounds lost by photolysis, microbial breakdown, volatilization, migration on soil particles through wind and water erosion, biological uptake and movement off-site, and atmospheric particulate movement should be identified.

Ideally, a comprehensive land treatment study will explain the fate of these compounds by examining kinetics, formation and degradation products. The use of solvents or other appropriate hydrogen-donating materials should be evaluated. Some work may require specific chemical analysis of 2,3,7,8-chlorinated congeners to insure that increased amounts of 2,3,7,8-chlorinated CDDs and CDFs relative to overall CDD and CDF levels are not formed. In short, if land treatment is a viable disposal technology, it will be so because CDDs and CDFs are destroyed during treatment rather than migrating to other environmental compartments. Because so many questions are unanswered, initial characterization of environmental fate during land treatment should be addressed by laboratory studies prior to full-scale field studies.

Much attention has been given to studying 2,3,7,8-tetraCDD because of its known mammalian toxicity. Kuehl et al. (1985b) examined the uptake and bioconcentration of all 22 possible tetraCDD isomers in carp fry during 30 day exposures to two types of incinerator fly ash, "east coast" and "midwest" (Table 3.2 provides fly ash composition.) Although other congeners were present in the fly ashes, only the tetraCDDs were looked for. Municipal incinerator fly ash is thought to be a source of 2,3,7,8-tetraCDD contamination in the Great Lakes watershed. In this "soup" type of exposure to mixtures of CDDs and CDFs, preferential accumulation and retention of 2,3,7,8-tetraCDD occurred. No other tetraCDDs were detected. This finding was observed in both static and flow-through tests and for 2,3,7,8-tetraCDD bound either to fly ash or released from the fly ash extract. The 2,3,7,8-tetraCDD was concentrated 100 times more from the extract than from the solid matrix. East Coast fly ash contained higher levels of 2,3,7,8-tetraCDD and four times more organic carbon than the midwest fly ash. However, carp concentrated 2,3,7,8-tetraCDD from the midwest fly ash to a greater extent than the east coast fly ash. The authors suggest that the availability of 2,3,7,8-tetraCDD may be inversely related to the amount of organic carbon present in the fly ash.

In a subsequent study that included additional CDD and CDF isomer groups, Kuehl et al. (1986a) exposed carp to fly ash for 60 days. The authors found that carp bioconcentrated not only 2,3,7,8-tetraCDD but also penta-, hexa-, and hepta- congeners of CDDs and CDFs chlorinated in the 2,3,7, and 8 positions (Table 3.3). In a related study, Kuehl et al. (1986b) exposed carp to sediment from a Wisconsin reservoir containing several CDDs and CDFs (Tables 3.4 and 3.5). A pattern of selective accumulation of 2,3,7,8-chlorinated congeners was observed, with the highest accumulation by 2,3,7,8-tetraCDD. Accumulation of only two non-2,3,7,8-chlorinated compounds from sediment, a pentaCDF and a hexaCDF, was detected.

Muir et al. (1985b) observed the bioconcentration of ^{14}C labelled 1,2,3,7-tetraCDD, 1,2,3,4,7-pentaCDD, 1,2,3,4,7,8-hexaCDD, and 1,2,3,4,6,7,8-heptaCDD in juvenile fathead minnows and rainbow trout by exposure to each of these congeners separately over a five day period. In a second study with a similiar exposure regime, Muir et al. (1986) exposed these same species to ^{14}C labelled 1,3,6,8-tetraCDD and octaCDD. The highest BCFs for isomers in both these studies averaged 4,232 for 1,2,3,4,7,8-hexaCDD and 5,702 for 1,3,6,8-tetraCDD for fathead minnows (Table 3.6). These BCFs are approximately one half the BCFs reported for 2,3,7,8-tetraCDD of 9,270 for rainbow trout (Branson et al., 1985).

Corbet et al. (1983) exposed fathead minnows and rainbow trout for 96 hours to 20 ng/l carbon 14-labelled 1,3,6,8-tetraCDD. They reported steady state BCF's of 610 and 210, respectively.

TABLE 3.1
2,3,7,8-TETRACDD BIOCONCENTRATION FACTORS FOR AQUATIC ORGANISMS

Species	2,3,7,8-TetraCDD Concentration in Water (ppt)[a]	Bioaccumulation/ Bioconcentration Factor[b]	Exposure Duration[c] (days)	Method	Concentration Initial/Final	Reference
Algae, Oedogonium cardiacum	2.42	2,075	7	Static	Final	Isensee, 1978 (Table 2)
Algae	0.05-239[d]	3,268[e] (avg.)	31	Static	Final	Isensee, 1978 (Table 1)
Pondweeds, Elodea nuttali & ceratophyllum emersum	53.7[f]	130 (max. conc.) (at 5 days)	5->60	Static (pond water)	Initial	Tsushimoto et al., 1982
Brine shrimp (Artemia salina)	100	1,570	4-7	Static	Final	Matsumura, 1977
Snail, Physa sp.	2.42	2,095	7	Static	Final	Isensee, 1978 (Table 2)
Snail	0.05-239[d]	6,106 (avg.)[e]	31	Static	Final	Isensee, 1978 (Table 1)
Mosquito larvae; (Aedes egypti)	450[g]	9,222	4-7	Static	Final	Matsumura, 1977
Daphnia magna	2.42	7,070	7	Static		Isensee, 1978 (Table 2)
	0.05-239[d]	4,438 (avg.)[e]	31	Static	Final	Isensee, 1978 (Table 1)
Channel catfish Ictalurus punctatus	0.05-239[d]	2,203 (avg.)	31	Static	Final	Isensee, 1978 (Table 1)
Mosquitofish Gambusia affinis	2.4	4,875	7	Static	Final	Yockim et al., 1978
	0.05-239[d]	6,970 (avg.)[e]	3	Static	Final	Isensee, 1978 (Table 1)
	2.42	4,850	7	Static	Final	Isensee, 1978 (Table 2)
Rainbow Trout	107	9270	6 hours	Static	Final	Branson et al., 1985

a/ All concentrations were analytically determined except Tsushimoto et al., 1982.
b/ Based on ^{14}C count as ^{14}C tetraCDD whole body, average values, wet weight.
c/ Soil treated with 2,3,7,8-tetraCDD and added to an aquatic ecosystem except for Tsushimoto et al., 1982 where 2,3,7,8-tetraCDD was added directly to pond water.
d/ Bioaccumulation Ratios (BR) were averages of several experiments using concentrations ranging from .05 - .239 ng/l. One of the concentrations (1330 ng/l) was unacceptably greater than the solubility of 2,3,7,8-tetraCDD (200 ng/l). Its BR was not included in the averages.
e/ Bioaccumulation ratios-organisms were exposed with other organisms.
f/ Estimated value assuming a homogenous distribution in water.
g/ This water concentration is slightly greater than water solubility of 2,3,7,8-tetraCDD (200 ppt).

TABLE 3.2 (continued)

CDD CONCENTRATIONS IN EAST COAST AND MIDWEST FLY ASH
(Adapted from Kuehl et al., 1985b)

CDD	Midwest Fly Ash pg/g [a/]	East Coast Fly Ash pg/g [b/]
Heptachloro-**		
1234679	36,000	54,000
1234678	42,000	53,000
Total	78,000	107,000
Octachloro-**		
12346789	52,000	95,000
Organic Carbon	1%	4%

* corrected for percent recovery of $^{13}C_{12}$ 2,3,7,8-tetraCDD
** absolute values not corrected for recovery
a/ from a midwestern municipal incinerator
b/ a blend of fly ash from 5 different municipal incinerators from the east coast.

TABLE 3.2

CDD CONCENTRATIONS IN EAST COAST AND MIDWEST FLY ASH
(Adapted from Kuehl et al., 1985b)

CDD	Midwest Fly Ash pg/g [a/]	East Coast Fly Ash pg/g [b/]
Tetrachloro-*		
2378	160	2,000
1469	55	600
1269	180	2,400
1267	86	1,500
1289	72	1,600
1369	970	5,200
1247 + 1248	2,200	19,000
1278	860	6,800
1268	1,000	8,900
1237 + 1238	3,600	26,000
1279	280	5,500
1246 + 1249	210	3,500
1478	180	2,300
1236	290	3,500
1239	250	3,700
1246 + 1249	210	2,100
1368	17,000	48,000
1379	13,000	45,000
1378	2,200	21,000
1234	550	16,000
Total	43,353	224,900
Hexachloro-**		
124679 + 124689	8,800	15,000
123468	46,000	35,000
123679 + 123689	30,000	32,000
123469	-	3,700
123478 + 123678	11,000	15,000
123467 + 123789	8,000	14,800
Total	103,800	165,600

TABLE 3.4

RESULTS OF CDD ANALYSIS OF WISCONSIN RESERVOIR SEDIMENT AND FISH
(Adapted from Kuehl et al., 1986b)

CONGENER	CONCENTRATIONS	
	Sediment (pg/g)	Carp (pg/g)
TetraCDD		
1,3,5,8-	17	ND**
*2,3,7,8-	170	120
PentaCDD		
1,2,4,6,8-; 1,2,4,7,9-	136	ND
1,2,3,6,8-	53	ND
1,2,4,7,8-	36	ND
1,2,3,7,9-	15	ND
1,2,3,4,7-; 1,2,4,6,9-	53	ND
*1,2,3,7,8-	31	4.8
1,2,3,6,9-	14	ND
1,2,4,6,7-; 1,2,4,8,9-	23	ND
1,2,3,6,7-	11	ND
1,2,3,8,9-	5	ND
HexaCDD		
1,2,4,7,9-; 1,2,4,6,8,9-; 1,2,3,4,6,8-	1090	ND
1,2,3,6,7,9-; 1,2,3,6,8,9-	580	ND
*1,2,3,6,7,8-	180	16
1,2,3,4,6,9-	16	ND
*1,2,3,7,8,9-	60	ND
HeptaCDD		
*1,2,3,4,6,7,8-	2190	27
1,2,3,4,6,7,9-	4720	ND
OctaCDD		
*1,2,3,4,6,7,8,9-	20,560	25

* Chlorinated at the 2,3,7, and 8 positions
** ND not detected; minimum level of detection, 1 pg/g

TABLE 3.3

CONCENTRATIONS OF CDDs AND CDFs IN CARP TISSUE AFTER 60 DAY EXPOSURE TO FLY ASH CONTAINING VARIOUS CDDs AND CDFs (Adapted from Kuehl, 1985a)

		Fly Ash[a/] (pg/g)	Carp (pg/g)
	1,3,6,8-tetraCDD	48,000	ND [b/]
	1,3,7,9-tetraCDD	45,000	ND
*	2,3,7,8-tetraCDD	2,000	7.5
*	1,2,3,7,8-pentaCDD	-	43
*	1,2,3,6,7,8/1,2,3,4,7,8-hexaCDD	15,800	105
*	1,2,3,4,6,7,8-heptaCDD	53,000	104
	1,2,3,4,6,7,9-heptaCDD	54,000	2
*	2,3,7,8-tetraCDF	-	7.2
*	1,2,3,7,8-pentaCDF	-	14
*	1,2,3,6,7,8-hexaCDF	-	24
*	1,2,3,4,6,7,8-heptaCDF	-	27

a/ A blend of flyash from 5 different incinerators on the east coast with 4 percent organic carbon. Dashes signify that no chemical analyses were performed

b/ ND = not detected

* Congeners chlorinated in the 2,3,7, and 8 positions

TABLE 3.5 (continued)

RESULTS OF CDF ANALYSIS OF WISCONSIN RESERVOIR SEDIMENT AND FISH
(Adapted from Kuehl et al., 1986b)

CONGENER	CONCENTRATIONS	
	Sediment (pg/g)	Carp (pg/g)
HexaCDF		
1,2,3,4,6,8-	21	ND
1,3,4,6,7,8-	91	ND
1,2,3,4,7,9-; 1,2,3,4,7,8-	30	ND
*1,2,3,6,7,8-	11	1
1,2,3,4,6,7-	84	2
1,2,3,6,8,9-; 1,2,3,4,8,9-	6	ND
HeptaCDF		
*1,2,3,4,6,7,8-	290	2.5
1,2,3,4,6,8,9-	430	ND
OctaCDF		
1,2,3,4,6,7,8,9-	850	ND(5)***

* Chlorinated at the 2, 3, 7, and 8 positions
** ND not detected, minimum level of detection, 1 pg/g
*** isomer detected but analysis did not meet quality assurance criteria at 5 pg/g

TABLE 3.5

RESULTS OF CDF ANALYSIS OF WISCONSIN RESERVOIR SEDIMENT AND FISH
(Adapted from Kuehl et al., 1986b)

CONGENER	CONCENTRATIONS	
	Sediment (pg/g)	Carp (pg/g)
TetraCDF		
1,3,7,8-	6	ND **
1,3,4,6-; 1,2,4,8-	20	ND
1,2,4,6-	14	ND
1,2,3,7-; 1,2,6,8-: 1,4,7,8-: 1,3,6,9-	15	ND
1,2,3,8-; 1,4,6,7-: 2,4,6,8-: 1,2,3,6-	31	ND
1,2,7,8-	88	ND
1,2,6,7-; 1,2,7,9-	10	ND
1,2,4,9-; 2,3,6,8-	19	ND
2,4,6,7-	7	ND
*2,3,7,8-	182	28
2,3,6,7-	24	ND
3,4,6,7-	5	ND
1,2,8,9-	8	ND
PentaCDF		
1,2,4,6,8-	64	ND
1,2,3,6,8-; 1,3,4,7,9-	9	ND
1,2,4,7,8-	22	ND
1,2,4,7,9-; 1,3,4,6,7-	3	ND
1,2,4,6,7-	8	ND
1,2,3,4,7-; 2,3,4,6,9-	4	ND
*1,2,3,4,8-; 1,2,3,7,8-	14	2.6
2,3,4,6,8-; 1,2,4,6,9-	9	ND
2,3,4,8,9-	6	ND
1,2,4,8,9-	5	ND
*2,3,4,7,8-	8	4.4
1,2,3,8,9-	2	ND
2,3,4,6,7-	2	2.8

Thus, it appears that non-2,3,7,8-chlorinated congeners can be bioconcentrated in aquatic organisms.

Two studies have elucidated target tissues for 2,3,7,8-tetraCDD uptake and retention. Kuehl et al. (1986b) examined the distribution of 2,3,7,8-tetraCDD in several specific tissues of male and female carp taken from a contaminated Wisconsin reservoir. Fillet, liver, visceral fat, brain, and cranial fat were analyzed. There appeared to be a greater deposit of 2,3,7,8-tetraCDD in fatty tissues, especially in cranial fat (Table 3.7).

TABLE 3.7

ORGAN-SPECIFIC 2,3,7,8-TETRACDD ANALYSIS OF MALE AND FEMALE CARP (Adapted from Kuehl et al., 1986b)

ORGAN	MALE (n=7) (pg/g)*	FEMALE (n=5) (pg/g)*
Fillet	23	28
Liver	93	150
Visceral Fat	280	300
Brain	68	24
Cranial Fat	370	370

*Detection limit: 1 pg/g.

The distribution of radiolabelled 2,3,7,8-tetraCDD fed 494 ng/kg/day to juvenile rainbow trout and yellow perch was determined after 13 weeks exposure (Kleeman et al., 1986a, 1986b). Rainbow trout had high concentrations in visceral fat, pyloric caeca, and the carcass; the carcass had the highest lipid concentration (Table 3.8). In yellow perch the visceral fat and liver had high concentrations of 2,3,7,8-tetraCDD but lipid concentrations for these were not given (Table 3.9).

Several of the bioconcentration studies reviewed have used contaminated sediment as the method of exposure. In these types of tests the BCF usually is based on the compound's concentration in water after its desorption from sediment. This experimental design was thought to prevent the compound's concentration in water from exceeding its solubility. However, for highly

TABLE 3.6

BIOCONCENTRATION OF CDD COMPOUNDS OTHER THAN 2,3,7,8-TETRACDD IN RAINBOW TROUT FRY AND FATHEAD MINNOWS (Adapted from Muir et al., 1985b and 1986)

Compound	Experiment Number	Conc.[a] in Water ng/l	BCF[b]
Rainbow Trout			
1,3,6,8-tetraCDD	1	4	2938 ± 480
	2	74	1964 ± 223
	3	211	1400 ± 473
1,2,3,7-tetraCDD	1	134	874 ± 83
	2	54	1577 ± 24
1,2,3,4,7-pentaCDD	1	16	810 ± 20
1,2,3,4,7,8-hexaCDD	1	47	1715 ± 112
	2	10	2840 ± 331
1,2,3,4,6,7,8-heptaCDD	1	55	1059 ± 91
	2	11	1790 ± 353
OctaCDD	1	415	34 + 18
	2	20	136 ± 55
Fathead Minnows			
1,3,6,8-tetraCDD	1	10	5840 ± 2859
	2	41	5565 ± 1550
1,2,3,7-tetraCDD	1	23	2018 ± 1
	2	28	2458 ± 206
1,2,3,4,7-pentaCDD	1	19	1647 ± 361
	2	11	1220 ± 157
1,2,3,4,7,8-hexaCDD	1	18	2630 ± 130
	2	7	5834 ± 1038
1,2,3,4,6,7,8-heptaCDD	1	39	513 ± 46
	2	8	515 ± 167
OctaCDD	1	9	2226 ± 1067

a/ Time-weighted average centrifuged water concentration during uptake.

b/ Bioconcentration factors are plus or minus the standard deviation.

TABLE 3.9

2,3,7,8-TETRACDD
CONCENTRATION AND LIPID CONCENTRATION
IN ORGANS OF YELLOW PERCH[a/]
(Adapted from Kleeman et al., 1986b)

ORGAN	2,3,7,8-TETRACDD[b/] CONCENTRATION	LIPID CONCENTRATION (g lipid/g tissue)
Visceral fat	2769 ± 134	
Liver	466 ± 33	
Spleen	166 ± 57	
Gill	155 ± 16	
Gastronintestinal tract	148 ± 19	
Pyloric caeca	143 ± 6	
Carcass	129 ± 7	0.19 ± 0.03
Kidney	119 ± 28	
Heart	77 ± 9	
Skin	41 ± 5	0.03[c/]
Skeletal muscle	9 ± 1	0.05 ± 0.01

a/ Yellow perch were killed at week 13 for analysis of 2,3,7,8-tetraCDD and lipid concentrations. Values are mean ± SE of six fish.

b/ pg equivalent concentration of ^{3}H tetraCDD/g

c/ Analysis of lipid concentration of skin pooled from six fish.

lipophilic compounds, the presence of dissolved organic carbon in water can sometimes allow a compound's concentration to exceed its water solubility.

Muir et al. (1984) designed an experiment to assess the influence of chemical properties, sediment type, and species characteristics on the bioavailability of CDDs to aquatic insects. They exposed five species of burrowing and nonburrowing insects to sand and sandy silt sediments containing either radiolabelled 1,3,6,8-tetraCDD or octaCDD. Test animals were exposed either directly to sediments or indirectly in waters overlying sediments. BCFs were determined based on 96-hour exposure (Table 3.10).

Two observations were made concerning the chemical characteristics of these two congeners (1,3,6,8-tetraCDD and octaCDD) with respect to bioavailability. First, the test animals had much lower BCFs for 1,3,6,8-tetraCDD and octaCDD than predicted from their water solubilities and partition coefficients. The authors suggest, that for octaCDD, the lower BCF may be due to its extreme hydrophobicity resulting in strong adsorption to sediment and dissolved organic carbon and to poor absorption across biological membranes due to steric factors. Alternatively, low BCFs could result from overestimation of the concentration in test water due to possible association with dissolved organic carbon, a form less suitable for bioavailability. In this study, octaCDD was present in concentrations which, after centrifugation, greatly exceeded its solubility. This phenomenon of octaCDD concentration apparently exceeding its water solubility has also been noted in other studies with octaCDD (Bruggeman et al., 1984; and Muir et al., 1986). The authors suggest that overestimation could occur because the measured radioactivity in water could include degradation products; these are possibly more polar and therefore less efficiently accumulated by the insects. Analytical confirmation for specific congeners was not conducted.

The second observation was that 1,3,6,8-tetraCDD was concentrated to a higher degree than octaCDD by all non-burrowing and burrowing insects. One reason may be that the structure of 1,3,6,8-tetraCDD is similar to 2,3,7,8-tetraCDD, which has better uptake than other congeners due to its optimal steric configuration. In bioaccumulation studies of superlipophilic chemicals, Bruggeman et al. (1984) found negligible accumulation of octaCDD in fish from aqueous and oral exposures. The author suggested that uptake is hindered by particular structural or physico-chemical properties interfering with membrane transport, such as molecular mass or size.

TABLE 3.10

BIOCONCENTRATION FACTORS FOR BURROWING AND NON-BURROWING INSECTS EXPOSED TO CDD CONTAMINATED SEDIMENT
(Adapted from Muir et al., 1984)

Animal	Sediment Type	Water Concentration (ng/l)[1/] % Sorbed (in parentheses) (96 hr)	Conc. in Pore Water[3/] (96 hr)	96 hr BCF in Water or Sediment[2/] BCFw	BCFsw	BCFs
1,3,6,8-TCDD						
Chironomus[4/]	Sand	366 (45)	3,843	1,554 ± 249	4,135 ± 185 *	394 ± 18
	Silt	44 (80)	57	1,992 ± 92	4,682 ± 1,042 *	3,602 ± 802 *
Hexagenia[4/]	Sand	166 (70)	1,743	849 ± 254	5,291 ± 619 *	504 ± 59
	Silt	55 (96)	72	2,846 ± 983	5,399 ± 1,506 *	4,153 ± 1,158 *
Paragnetina[5/]	Sand	205 (58)	2,152	830 ± 69	1,048 ± 364	100 ± 35
	Silt	142 (34)	185	———	136 ± 20	105 ± 15
Acroneuria[5/]	Sand	" "	"	794 ± 211	963 ± 95 **	92 ± 9
	Silt	" "	"	———	182 ± 77	140 ± 59
Pteronarcys[5/]	Sand	" "	"	———	843 ± 157	80 ± 15
OCDD						
Chironomus[4/]	Sand	235 (71)	1,814	141 ± 104	183 ± 70	24 ± 9
	Silt	101 (81)	101	145 ± 95	77 ± 44	77 ± 44
Hexagenia[4/]	Sand	102 (67)	787	———	1,086 ± 465	141 ± 60
	Silt	24 (99)	24	———	1,019 ± 427	1,109 ± 427
Paragnetina[5/]	Sand	182 (36)	1,405	42 ± 13	81 ± 19 *	11 ± 2
	Silt	147 (41)	147	———	15 ± 2	15 ± 2
Acroneuria[5/]	Sand	" "	"	———	99 ± 38	13 ± 5
	Silt	" "	"	———	20 ± 7	20 ± 7
Pteronarcys[5/]	Sand	" "	"	160 ± 6	156 ± 45	20 ± 6

1/ = Water concentration after centrifugation at 20,000 g (30 min). Mean of duplicate samples.
2/ = BCFw = average concentration insects in water (96 hr)/average water conc. over interval;
BCFsw = average concentration insects in sediment (96 hr)/average water conc. and
BCFs = average concentration insects in sediment (96 hr)/pore water concentration.
3/ = Pore water concentration was determined on the supernatant of centrifuged wet sediment.
4/ = Chironomus and Hexagenia are burrowing insects (detritivores).
5/ = All water data for stonefly nymphs (Paragnetina, Acroneuria, and Pteronarcys) are combined (Means of 6 samples). These are non-burrowing insects.
* = Indicates significant difference between BCFw and BCFs or BCFsw at P = 0.01.
** = Indicates significant differences between BCFw and BCFs or BCFsw at P = 0.05.

Species characteristics and sediment type affected the bioaccumulation of 1,3,6,8-tetraCDD (Muir et al., 1984). The burrowing insects (detritivores) had higher BCFs for 1,3,6,8-tetraCDD than the non-burrowing insects for all types of exposures. BCFs were especially high for those detritivores exposed to a sediment containing silt. Ingestion of sediment during the burrowing activity of the detritivores, especially mixtures of sand and silt that contain a size range of particles favored by these animals, may explain their greater BCFs. These organisms also had come into contact with pore water (water within passageways in the sediment), which was generally more contaminated than the water above the sediment. This difference in BCFs between the burrowing insects and non-burrowing insects was not observed in any of the octaCDD exposures.

Isensee (1978) and Isensee and Jones (1975) exposed daphnids, mosquitofish, catfish, and snails to 2,3,7,8-tetraCDD-contaminated sediment and measured concentrations in sediment, tissue, and water. The bioconcentration factors averaged over a range of test concentrations for these organisms were 4,438, 6,970, 2,203, and 6,106, respectively. They found significant correlations ($r = 0.94$ to .97) between 2,3,7,8-tetraCDD concentrations in water and tissues for a wide range of water concentrations. The authors concluded that the amount of 2,3,7,8-tetraCDD accumulated by the organisms in this test was controlled almost entirely by the amount of 2,3,7,8-tetraCDD available in the water. However, correlations for sediment and tissue were not calculated and one might argue that catfish and snails ingest a significant amount of sedimentary material while feeding.

Kuehl et al. (1986b), as discussed above, reported levels of CDDs and CDFs in carp exposed to contaminated sediment. These fish, although not inhabitants of bottom mud or soil, were found in this study to contain large amounts of sediment in their intestines. The authors, in an attempt to determine whether these carp (exposed in the laboratory) had accumulated 2,3,7,8-tetraCDD from water across the gills, analyzed the bioassay water after centrifugation and did not find 2,3,7,8-tetraCDD. They contended that the rate of CDD desorption from sediment to water while passing over the gills is too slow to influence the amount of CDD available for uptake by that route. They concluded that the level of 2,3,7,8-tetraCDD detected was essentially from sediment that passed through the gut during the course of normal feeding behavior.

Bioconcentration In Aquatic Plants

Three studies have investigated bioconcentration of 2,3,7,8-tetraCDD by aquatic plants. Isensee and Jones (1975); and Isensee (1978) reported average bioaccumulation ratios of 3,260 and 2,075 for an alga, Oediogonium cardiacum after a 31 day exposure. Pondweeds, Elodea nuttali and Ceratophyllon demersum, reached a maximum bioconcentration factor of 130 after 5 days of a 50 day study in a manmade outdoor pond (Tsushimoto et al., 1982). Kenaga and Norris (1983) criticized reported bioconcen- values as being strongly affected by adsorption of 2,3,7,8-tetraCDD onto the surface of plants. The methods used in these studies to determine 2,3,7,8-tetraCDD concentrations did not consider adsorption.

Metabolism

To study the metabolism of 2,3,7,8-tetraCDD in fish, Kleeman et al. (1986a and 1986b) analyzed the liver, kidney, and skeletal muscle of adult yellow perch and rainbow trout one week after a single injection of 60 ug/kg radiolabelled 2,3,7,8-tetraCDD. In these tissues, the parent compound accounted for all of the extractable ^{14}C in rainbow trout and 96 to 99 percent in yellow perch. However, the gallbladder bile of both of these species contained the parent compound and 2,3,7,8-tetraCDD metabolites. At least one of the metabolites in the bile of both species was a glucuronide conjugate.

Depuration and Elimination

Water Exposure

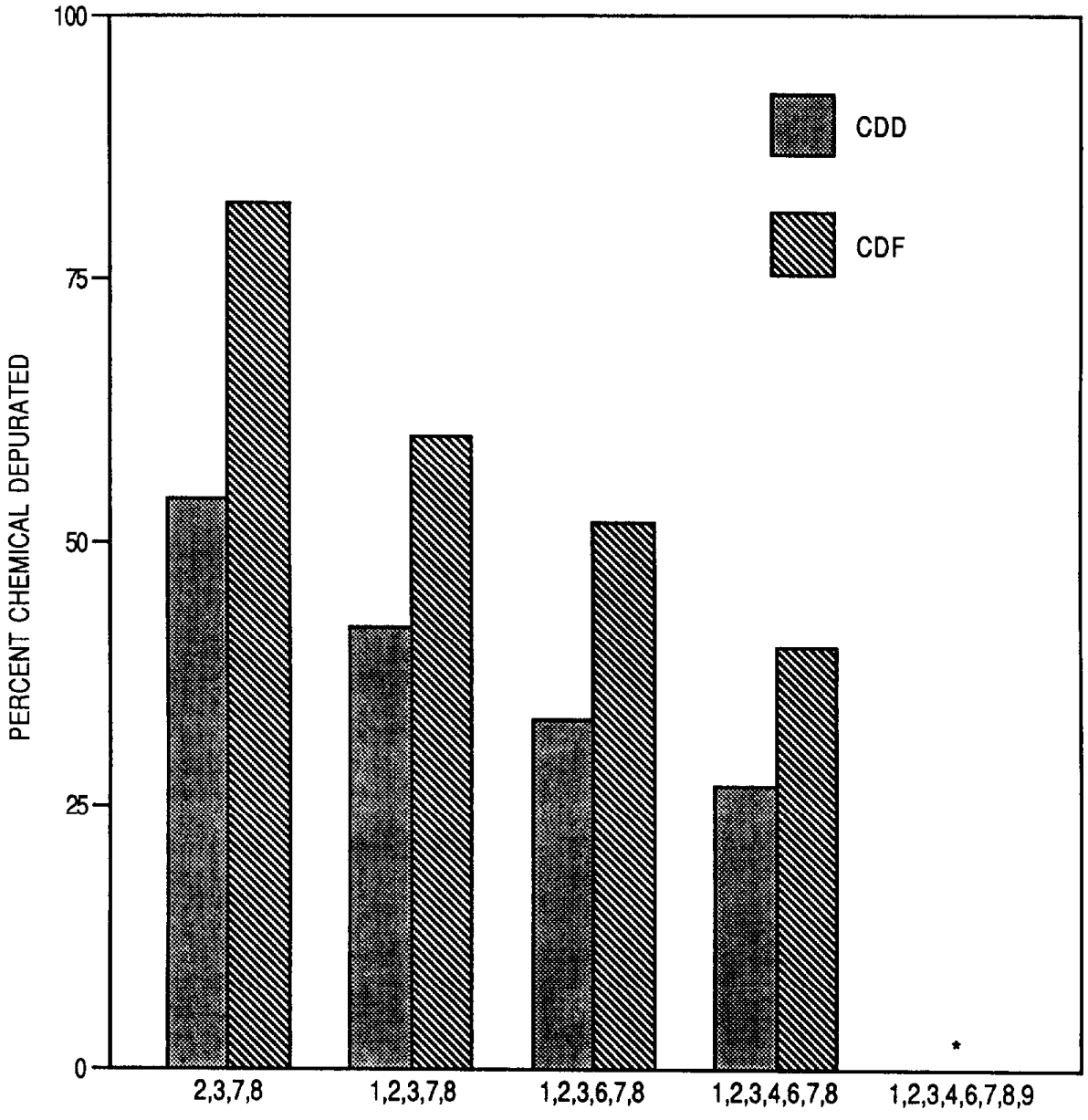
Kuehl et al. (1985a, 1986b) attempted to determine the accumulation and depuration of 2,3,7,8-tetraCDD in carp exposed to sediment containing 39 pg/g 2,3,7,8-tetraCDD. After 55 days of exposure, the carp (5 to 8 percent lipid) had accumulated 2,3,7,8-tetraCDD to a level of 7.5 pg/g; however, a steady state had not yet been reached. The fish were then placed in clean water to observe depuration. At 205 days only 33 percent depuration had occurred.

In the same study, carp (15 to 18 percent lipid) were taken from a contaminated reservoir in Wisconsin and maintained in clean water for 336 days. The carp lipid fraction was analyzed for 2,3,7,8-tetraCDD on days 1, 64, 119, 224, and 336. Other 2,3,7,8-chlorinated CDDs and CDFs were also analyzed for on days 1 and 336 (Figure 3.1 and Table 3.11). Depuration rates of both CDDs and CDFs decreased with increasing chlorination. For the same degree of chlorination, the percentage of CDFs depurated

Figure 3.1

DEPURATION OF 2,3,7,8-SUBSTITUTED CDDs AND CDFs FROM CARP AFTER 336 DAYS

(Adapted from Kuehl et al., 1985b)



* NO CHANGE IN CONCENTRATION DETECTED.

TABLE 3.11

DEPURATION OF 2,3,7,8-SUBSTITUTED CDDs AND CDFs FROM CARP AFTER 336 DAYS[a/]
(Data from Kuehl et al., 1985a)

Compound	Day 1	Day 336	Percent Depurated
CDD			
2,3,7,8-tetraCDD	370	170	54
1,2,3,7,8-pentaCDD	13	7.5	42
1,2,3,6,7,8-hexaCDD	24	16	33
1,2,3,4,6,7,8-heptaCDD	30	22	27
1,2,3,4,6,7,8,9-octaCDD	38	38	0
CDF			
2,3,7,8-tetraCDF	150	27	82
1,2,3,7,8-pentaCDF	4.9	1.9	60
1,2,3,6,7,8-hexaCDF	5.4	2.6	52
1,2,3,4,6,7,8-heptaCDF	5.3	3.2	40
1,2,3,4,6,7,8,9-octaCDF	12	12	0

a/ Expressed as pg/g lipid.

---

was greater than that for CDDs. No depuration of octaCDD or octaCDF was observed. The half-life of 2,3,7,8-tetraCDD in tissues was found to be approximately 300 to 320 days (Kuehl et al., 1985a, 1986b). Half-lives for CDF isomers in this study were not reported but tetra-, penta-, and hexaCDFs had depurated over 50 percent by day 336. Hepta- and octaCDFs had not reached 50 percent depuration.

The shortest depuration half-life reported for 2,3,7,8-tetraCDD is 58 days for rainbow trout after a six hour exposure to 107 ng/l 2,3,7,8-tetraCDD in water (Branson et al., 1985). This half-life is still far greater than any other isomers studied.

Muir et al. (1985b and 1986) observed the depuration half-lives in rainbow trout and fathead minnows exposed through water for five days to concentrations of six congeners from five different isomer groups (Table 3.12). The half-lives for the hexa- and

TABLE 3.12

HALF-LIVES OF CDD COMPOUNDS OTHER THAN 2,3,7,8-TETRACDD IN FISH
(Adapted from Muir et al., 1985b and 1986)

SPECIES AND CONGENER	AVERAGE HALFLIFE (days)
Rainbow Trout	
1,2,6,8-tetraCDD	7.5
1,2,3,7-tetraCDD	2.7
1,2,3,4,7-pentaCDD	2.5
1,2,3,4,7,8-hexaCDD	15
1,2,3,4,6,7,8-heptaCDD	16.7
OctaCDD	6
Fathead Minnow	
1,3,6,8-tetraCDD	7.5
1,2,3,7-tetraCDD	2.8
1,2,3,4,7-pentaCDD	3.2
1,2,3,4,6,7,8-hexaCDD	23.5
1,2,3,4,6,7,8-heptaCDD	17.5
OctaCDD	13

heptaCDDs were longer than for the lower chlorinated tetra- and pentaCDDs, which corresponds to higher octanol/water partition coefficients for more highly chlorinated CDDs. However, the elimination half-life of octaCDD in rainbow trout was almost equal to 1,3,6,8-tetraCDD. A decrease in the depuration of CDDs from carp as the degree of chlorination increased was observed by Kuehl et al. (1985a, 1986b) for 2,3,7, and 8 chlorinated congeners including octaCDD. The water concentration of octaCDD was much greater than its solubility. As a result, the authors suspect that the octaCDD was not absorbed by the fish, but adsorbed on their skin.

Food Exposure

In feeding studies no consistent relationship was found between half-lives and degree of chlorination when 2,7-diCDD, 2,3-diCDD, 1,2,3,4-tetraCDD, octaCDD, 3,6-diCDF, and octaCDF were fed to

rainbow trout in one single dose (Niimi and Oliver, 1986). Half lives in days for the CDDs and CDFs ranged from 2 to 43 and 7 to 12, respectively (Table 3.13). Absorption efficiencies were low, ranging from 2 to 16 percent. However, 2,7-diCDD, which has a structure similar to 2,3,7,8-tetraCDD, had greater than 30 percent assimilation, a finding that suggests absorption through the gut might be influenced by the steric configuration of these compounds.

In two other longer term feeding studies, Kleeman et al. (1986a and 1986b) exposed rainbow trout and yellow perch to a diet of 494 pg/g of radiolabelled 2,3,7,8-tetraCDD for 13 weeks and observed half-lives of 105 and 126 days, respectively.

ACUTE AND CHRONIC TOXICITY

According to EPA (1984a), available fish and invertebrate acute and chronic toxicity data for 2,3,7,8-tetraCDD are too limited to permit derivation of water quality criteria. However, the available studies can give useful indications of toxicity. The majority of acute and chronic toxicity investigations have studied only the toxicity of 2,3,7,8-tetraCDD to freshwater species. Most studies have been static or static renewal bioassays. Only one acute bioassay has been reported involving CDDs and CDFs other than 2,3,7,8-tetraCDD. However, this test included exposure to several congeners simultaneously, and it is unclear which congener caused the reported toxicity. Few chronic toxicity studies exist from exposure to CDDs and CDFs. Polychlorinated dibenzofurans mainly have been studied in terms of chronic oral toxicity (e.g., feeding studies).

Acute Toxicity

Fish: Delayed Effects

A majority of the reported 2,3,7,8-tetraCDD acute toxicity studies on aquatic organisms have shown a pattern of delayed effects, mimicking the expected response time of low level, long term exposures. This same unusual action pattern has also been observed in acute exposures of 2,3,7,8-tetraCDD to mammals (McConnell et al., 1978). Due to this delay, 96-hour acute tests with 2,3,7,8-tetraCDD are typically followed by long observation periods of up to 24 weeks.

Miller et al. (1973) observed in 96 hour static exposure tests with coho salmon and guppies that initial responses did not occur for 5 to 10 days after the exposure period and mortality often extended over the next 2 months. Helder (1981) exposed juvenile

TABLE 3.13

HALF-LIVES OF CDDs IN RAINBOW TROUT
AFTER A SINGLE ORAL EXPOSURE
(Adapted from Niimi and Oliver, 1986)

COMPOUND	ORAL EXPOSURE CONCENTRATION (ug/l oil)	HALF-LIFE (Whole Body) (Days)
2,7-diCDD	82	2
2,3-diCDD	37	7
1,2,4-triCDD	38	12
1,2,3,4-tetraCDD	30	43
octaCDD	30	15
3,6-diCDF	115	12
octaCDF	15	7

rainbow trout to 100 ppt 2,3,7,8-tetraCDD for 96 hours and observed a sudden increase in mortality after the 21st day; by the 27th day, all trout had died.

Helder (1980 and 1981) exposed rainbow trout and pike eggs to concentrations of 0.1 to 10.0 ng/l 2,3,7,8-tetraCDD for 96 hours. Observation for mortality continued through three life stages: egg, yolk sac fry, and feeding fry. For both species, significant mortality occurred in the yolk sac fry stage at 1.0 and 10.0 ng/l (Table 3.14). The author suggested that the highly lipophilic character of 2,3,7,8-tetraCDD may be the cause of the high mortality to yolk sac fry. This stage may be vulnerable because, as demonstrated *in vitro* by other lipophilic compounds, 2,3,7,8-tetraCDD may readily accumulate in the triglyceride fraction of the yolk and become mobile several days after hatching when the fry utilize the triglycerides in the yolk as food.

TABLE 3.14

PERCENT MORTALITY OF RAINBOW TROUT AND NORTHERN PIKE FRY WITH YOLK SAC EXPOSED TO 2,3,7,8-TETRACDD
(Data from Helder, 1980 and 1981)

	EXPOSURE CONCENTRATIONS (ng/l)				
	Acetone Control	Control	0.1	1.0	10.0
Rainbow trout	0.9	0.8	0.9	2.3[a/]	15.8[a/]
Northern pike	14.0	9.2	14.2	48.8[a/]	94.1[a/]

[a/] $p>0.001$

Due to the delayed lethality normally found in 2,3,7,8-tetraCDD bioassays, the expression of LC_{50} values as the concentration of toxicant giving 50 percent mortality at the end of a 96 hour exposure is not a meaningful indicator of 2,3,7,8-tetraCDD toxicity. As a result, the literature reports modified LC_{50}s, measuring mortality at some given time after the exposure period. There is not yet agreement on a standardized post-exposure delay for the calculation of LC_{50}. Among 2,3,7,8-tetraCDD modified LC_{50} values reported for 96 hour exposure tests on fish are 100 ng/l at 21 days for juvenile rainbow trout (Helder, 1981) and 5.6 ug/l (LC_{55}) at 60 days for juvenile coho salmon (Miller et al., 1979). See Table 3.15 for a summary of 2,3,7,8-tetraCDD bioassays. These data are presented in Figure 3.2.

TABLE 3.15

EFFECTS OF 2,3,7,8-TETRACDD ON AQUATIC ORGANISMS
(Adapted from Kenaga and Norris, 1983)

SPECIES	TEST DURATION[a/]	EFFECT	CONC. IN WATER (ng/l)	REFERENCE
Snail				
Physa sp.				
(adult)	36d/48d	reduced reproduction	200	Miller et al., 1973
Oligochaete worm				
Paranais sp.				
(adult)	55d/55d	reduced reproduction	200	" " "
Mosquito				
Aedes aegypti				
(larvae)	17d/39d	No effect on pupation	200	" " "
Guppy				
Poecilia reticulat				
(9 - 40mm)	5d/37d	Feeding decline, skin discoloration, fin necrosis, mortality	10000	" " "
"	5d/5d	LC8	100	Norris & Miller, 1974
" " "	5d/21.7d	LC50	100	"
" " "	5d/11.6d	LC50	1000	"
" " "	5d/18.2d	LC50	10000	"
" " "	5d/37d	LC100	100	"
Coho salmon				
Oncorhynchus kisutch				
(juvenile)	24-96h/40d	LC100	56	Miller et al., 1973
"	96h/60d	LC55	5.6	" " "
"	96h/60d	No effect on feeding, growth, and survival	0.56	Miller et al., 1979
Mosquitofish				
Gambusia affinis				
(gravid)	8d/15d	LC100	2.4-4.2	Yockim et al., 1978

TABLE 3.15 (continued)

EFFECTS OF 2,3,7,8-TETRACDD ON AQUATIC ORGANISMS
(Adapted from Kenaga and Norris, 1983)

SPECIES	TEST DURATION[a/]	EFFECT	CONC. IN WATER (ng/l)	REFERENCE
Northern pike				
Esox lucius				
(eggs)	96h/23d	LC26	0.1	Helder, 1982
"	96h/23d	LC53	1	" "
"	96h/23	LC99	10	" "
Rainbow trout				
Salmo gairdneri				
(juvenile)	64h/72d	LC12	10	Helder, 1981
"	64h/21d	LC50	100	" "
"	64h/27d	LC100	100	" "
"	64h/73d	EC54 - growth	10	" "
American bullfrog				
Rana catesbeiana				
adults [b/]	-- /35d	No mortality	500[c/]	Beatty et al., 1976
tadpoles	"	" "	1000[c/]	" " " "
Alga				
Oedogonium				
cardiacum	32d/32d	No effect	1330	Isensee, 1978

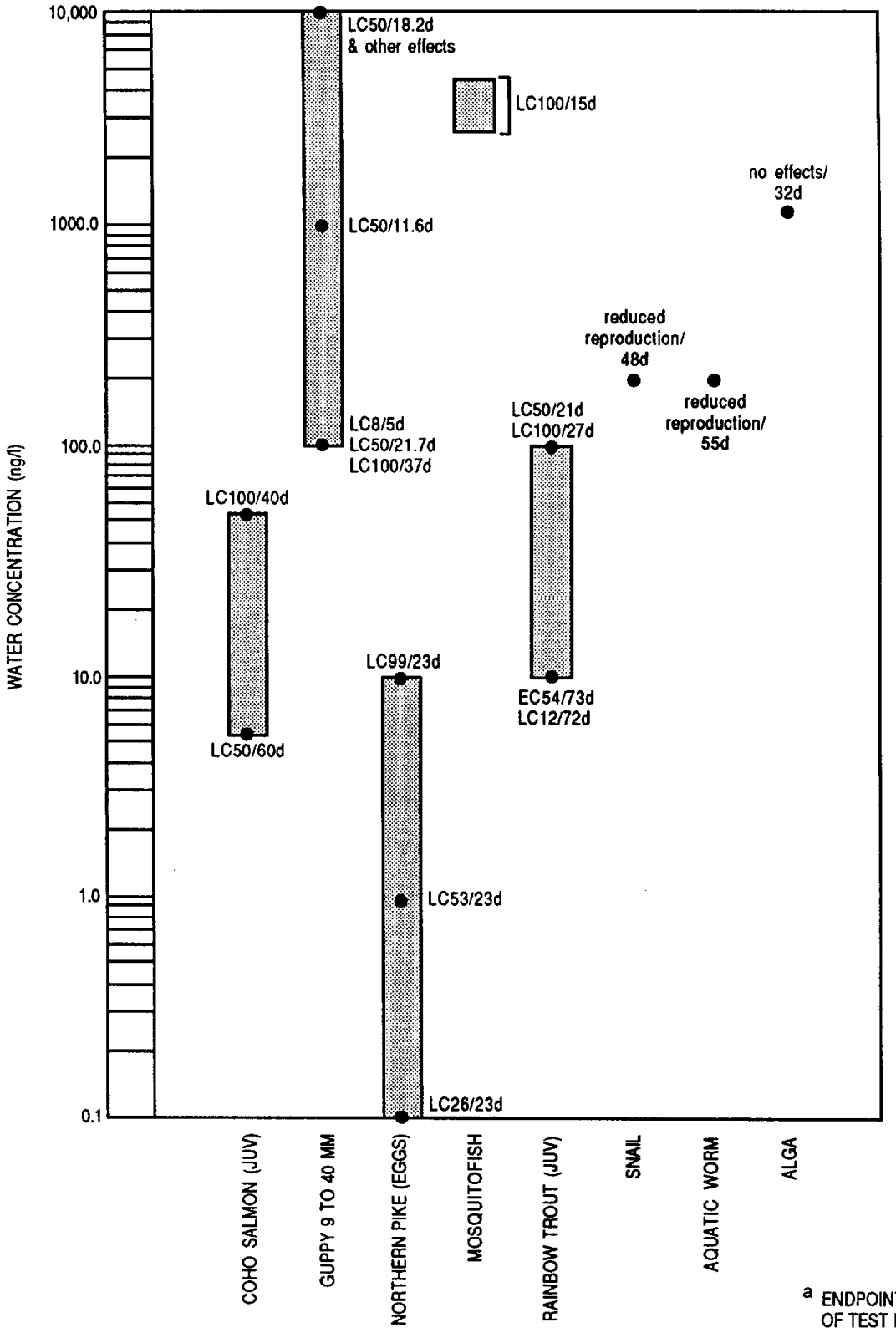
a/ Duration of exposure/post exposure observation period

b/ One intraperitoneal injection

c/ Dosage expressed in ug/kg of organism or food

Figure 3.2

RANGE OF 2,3,7,8-TETRACDD CONCENTRATIONS TOXIC TO AQUATIC SPECIES [a]



[a] ENDPOINT/DURATION OF TEST IN DAYS

Fish: Growth Effects

The most common effect reported in the 2,3,7,8-tetraCDD bioassay literature was growth retardation for several species of fish. For rainbow trout yolk sac fry exposed as eggs to 0.1, 1.0, and 10 ng/l of 2,3,7,8-tetraCDD for 96 hours, significant growth retardation occurred at all levels of exposure (Figure 3.3). At the 0.1 and 1.0 ng/l exposure concentrations, reduced growth was not significant until 72 and 118 days, respectively, after fertilization (exposure began just after fertilization). However, at the 10 ng/l concentration, growth retardation occurred throughout the entire experiment (Helder, 1981). In the same study, yolk sac fry were also exposed to 2,3,7,8-tetraCDD at 1.0 ng/l and juveniles at 10 and 100 ng/l. Significant growth retardation occurred at all concentrations during the entire experiment. Pike fry exposed to 2,3,7,8-tetraCDD in the egg stage for 96 hours to 0.1, 1.0, and 10 ng/l showed significantly shortened body lengths for a long period of time after hatching (Helder, 1980) (Figure 3.4). The growth of coho salmon was markedly inhibited by the 80th day after a 96-hour exposure to 56 ng/l 2,3,7,8-tetraCDD (Miller et al., 1973) (Figure 3.5).

Fish: Histopathology

Several acute toxicity studies on fish have found histopathological effects such as fin necrosis, loss or underdevelopment of caudal fins, edema, liver necrosis, and hemorrhaging from exposures to 2,3,7,8-tetraCDD ranging from 0.1 ng/l to 10 ug/l (Norris and Miller, 1974; Helder, 1980, 1981 and 1982; Miller et al., 1973 and 1979). Edema, often generalized, was the most consistent syndrome among several species.

Helder (1980 and 1981) conducted extensive studies on the histopathological effects of 2,3,7,8-tetraCDD on rainbow trout eggs, yolk sac fry and juveniles and on pike eggs. For the eggs of both species exposed to 1.0 and 10 ng/l of 2,3,7,8-tetraCDD, generalized hemorrhaging and edema increased with increasing dose. Degenerative changes and necrosis in the liver parenchymal cells were also observed. Newly hatched rainbow trout yolk sac fry exposed to 1 ng/l 2,3,7,8-tetraCDD were affected in the same manner. The exposed rainbow trout eggs that survived to the fry stage (12 weeks) developed shortened maxillas and opercular defects.

Effects of 2,3,7,8-tetraCDD on Growth of Fish

Figure 3.3
EFFECTS ON RAINBOW TROUT WEIGHT
AFTER 96 HR. EXPOSURE TO 2,3,7,8-TetraCDD
(Adapted from Helder, 1981)

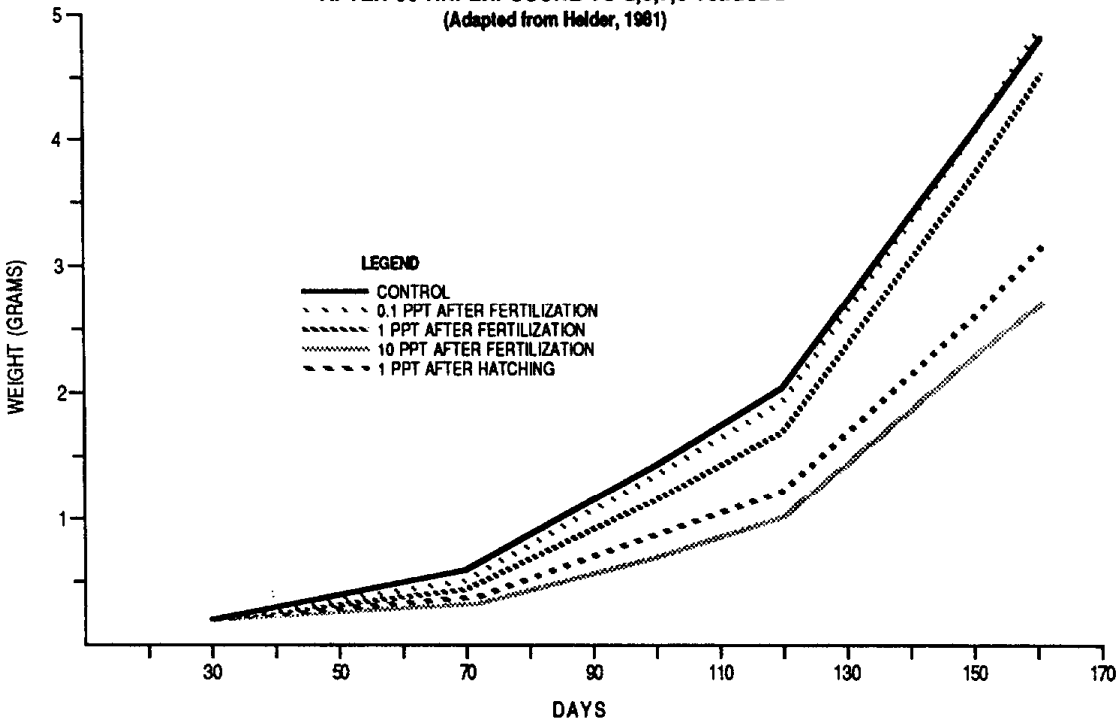


Figure 3.4
EFFECTS ON PIKE BODY LENGTH
AFTER 96 HR. EXPOSURE TO 2,3,7,8-TetraCDD
(Adapted from Helder, 1980)

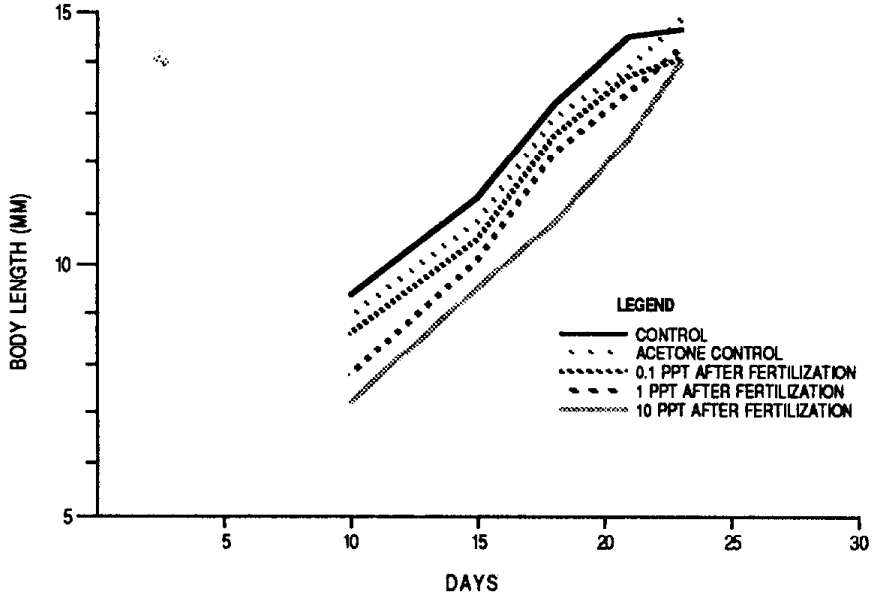
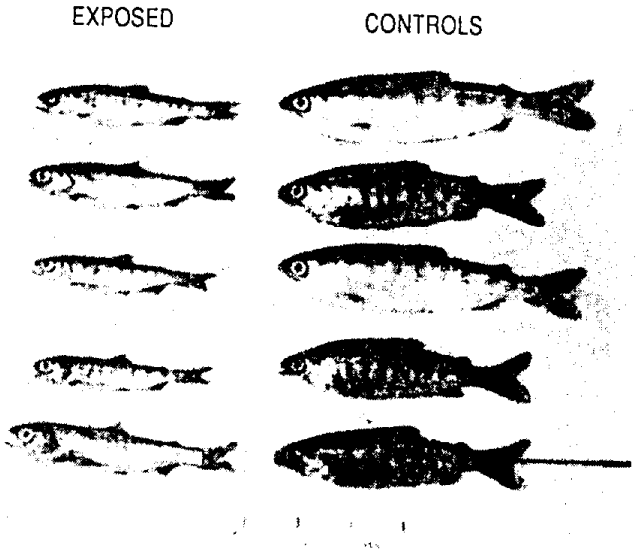


Figure 3.5

EFFECTS ON COHO SALMON GROWTH AT 80 DAYS
AFTER A 96 HR. EXPOSURE TO 56 ng/l OF 2,3,7,8-TetraCDD

(Miller et al.,1973)



Invertebrates

According to EPA (1984a), there are no data available to calculate 48 or 96 hour LC_{50} or EC_{50} values for invertebrate exposures to any CDD or CDF.

Amphibians

One study examined the effects of 2,3,7,8-tetraCDD on the larval and adult American bullfrog, Rana catesbeiana (Beatty et al., 1976). Intraperitoneal injections of up to 1 mg 2,3,7,8-tetraCDD/kg of body weight showed no effect on either lifestage during 35 observation days. Although this method of toxicant administration is not comparable to published studies on fish, the study suggests that Rana may be less sensitive to 2,3,7,8-tetraCDD.

Chronic Toxicity

Fish

Few chronic toxicity studies from exposure through the water medium are reported in the literature. Oral chronic toxicity studies have been conducted with 2,3,7,8-tetraCDD on rainbow trout, and with chlorinated dibenzofurans on Atlantic salmon and brook trout.

In a study by Yockim (1978) to observe the toxicity and environmental fate of 2,3,7,8-tetraCDD, mosquitofish (Gambusia affinis) were exposed to 2,3,7,8-tetraCDD concentrations in water ranging from 2.4 to 4.2 ng/l. All fish died after 15 days exposure. Gravid mosquitofish exposed to the same concentrations for 8 days lost all external signs of pregnancy and died after 15 days. The gravid control fish remained visibly pregnant and at autopsy showed fry in the late stages of development.

Hawkes and Norris (1977) fed young rainbow trout 2,3,7,8-tetraCDD in dried fish food at levels of 2.3 ppt, 2.3 ppb, 2.3 ppm, equivalent to an intake level of 0.0000064, 0.0072, and 4.2 ug 2,3,7,8-tetraCDD/kg body wet weight/day, respectively (calculated by Kenaga and Norris (1983) assuming wet weight is 5 times dry weight). Levels of 0.0000064 and 0.0072 ug/kg had no effect on food consumption, growth, or survival. However, when fish were exposed to 4.2 ug/kg, 50 percent mortality occurred after 61 days and 96 percent mortality by 71 days. At 4.2 ug/kg other effects were observed, including decreased feeding, growth reduction, fin erosion, and changes in liver tissue. These effects are quite similiar to those seen in fish exposed to 2,3,7,8-tetraCDD in water.

There is little information on the chronic toxicity of specific higher chlorinated dibenzofurans. Currently available data include very brief reports on chronic toxicity studies of fish fed CDF contaminated food.

Zitko and Choi (1973) reported that juvenile Atlantic salmon fed dry fish food spiked with mixed di-, tri-, tetra-, and octaCDFs in concentrations of 2.1, 4.4, 2.2, and 9.7 ppm respectively, for up to 140 days showed median mortality at 120±30 days. Only octaCDF was found in the tissues of the salmon.

However, Zitko et al., (1973) found no mortality when immature brook trout were fed several doses of 2,8-diCDF totaling 107 to 361 ug/g wet weight for 50 days. No mortality resulted, even after administration of a single dose as high as 122 ug/g.

Invertebrates

Miller et al. (1973) conducted long term static bioassays on snails (Physa sp.), mosquito larvae (Aedes aegypti), and aquatic worms (Paranais sp.). At exposures of 0.2 ug/l 2,3,7,8-tetraCDD, the mosquito larvae were not affected, but snails and worms both showed reduced reproductive success.

Aquatic Plants

Aquatic plants appear to be insensitive to low concentrations of 2,3,7,8-tetraCDD. No attempts have been made to determine the maximum no-effect levels (Kenaga and Norris, 1983). The limited existing data are from microcosm studies in which an alga, Oedogonium cardiacum, was not affected in a 31 day exposure to 2,3,7,8-tetraCDD concentrations ranging from 2.4 to 4.2 ng/l (Yockim et al. 1978). In a separate study, O. cardiacum was not affected in higher 2,3,7,8-tetraCDD concentrations of up to 1330 ng/l for a 31 day exposure (Isensee and Jones, 1975; Isensee, 1978).

Mechanisms of Action

Information on CDD and CDF mechanisms of action is lacking. A few, very general and brief, discussions focus only on the 2,3,7,8-tetraCDD congener. In a discussion on the sites and mechanisms of toxicity of 2,3,7,8-tetraCDD, Norris and Miller (1974) noted that delayed mortality in guppies is consistent with the hypothesis that 2,3,7,8-tetraCDD induces liver dysfunction; such dysfunction has been shown in tests with rodents. However, Helder (1982) suggests that his observation of hemorrhaging and

edema within the eggs of rainbow trout 21 days after exposure to 2,3,7,8-tetraCDD indicates a mechanism other than hepatic damage, because the liver at this point is just beginning to develop. He suggests the damage in this case is probably vascular.

SUMMARY AND DISCUSSION

Bioconcentration

Bioconcentration factors have not been reported in the literature for chlorinated dibenzofurans. Measured BCFs for 2,3,7,8-tetraCDD and other chlorinated dibenzo-p-dioxins are lower than would be expected from their high K_{ow} (octanol/water partition coefficient) values. However, most of the bioconcentration and bioaccumulation studies reviewed did not determine BCFs after a steady state had been reached. Thus, short-term laboratory exposures may give underestimates of potential BCFs for organisms exposed in the environment for long periods of time. Limited membrane transport of CDDs also may result in lower BCFs than expected. This limitation could be due in part to their large molecular size, high K_{ow} values and low water solubilities, resulting in binding to particulates and dissolved organic carbon (DOC). Congeners associated with particulates or dissolved organic carbon tend to show concentrations in the water higher than what may actually be available to non-filter feeding organisms.

Many of the studies reviewed used radiolabelled ^{14}C CDDs to determine bioconcentration factors. This method could result in overestimation of CDD concentrations in water and therefore lower BCFs. This is because the measured radioactivity may include degradation products if actual chemical species are not analytically identified.

Steric configurations such as the planar structure of 2,3,7,8-tetraCDD can affect the rate of membrane transport. The BCF for 2,3,7,8-tetraCDD is higher than that for any other congener. Fish and invertebrates have been shown to concentrate 2,3,7,8-tetraCDCD in their tissues up to approximately 9000 times the concentration in water. Aquatic organisms exposed to mixtures of many different CDDs and CDFs in water tend to accumulate 2,3,7,8-chlorinated congeners. However, non-2,3,7,8-chlorinated congeners, in the absence of 2,3,7,8-chlorinated congeners, can be accumulated in aquatic organisms.

Distribution of 2,3,7,8-tetraCDD in fish tissue follows what would be expected from its high octanol to water partition coefficient. The greatest proportion of the accumulated 2,3,7,8-tetraCDD is associated with the fatty tissue of aquatic organisms exposed to contaminated water.

Compared with research using rodents and mammals on the metabolism of CDDs, very little is known about CDD metabolism in aquatic species. Glucuronide conjugates found in the bile of yellow perch and rainbow trout indicate biotransformation, but the mechanism is not known.

Long-term water exposures approaching equilibrium show that the retention time of 2,3,7,8-chlorinated congeners in tissues of aquatic organisms increases with increasing chlorination. The effect that longer retention of more highly chlorinated congeners has on toxicity is uncertain due to the lack of aquatic toxicity data for congeners other that 2,3,7,8-tetraCDD.

Toxicity

Two unusual aspects of 2,3,7,8-tetraCDD's toxicity which make it unique are (1) its pattern of delayed effects after acute exposures and (2) the inordinately low concentrations (as low as 0.1 ng/l) which cause a toxic reaction in aquatic organisms.

Growth retardation was the most commonly reported effect of several species of fish yolk sac fry after 96-hour exposures to 2,3,7,8-tetraCDD. Histopathological effects included fin necrosis, edema, liver necrosis, and hemorrhaging. Edema, the most consistently reported syndrome, may be the result of induced liver dysfunction or vascular damage observed in fish exposed to 2,3,7,8-tetraCDD.

Aquatic plants tested at low exposures appear to be insensitive to 2,3,7,8-tetraCDD. The effects of other CDD and CDF congeners on aquatic organisms are unknown. Studies are needed to determine toxicity of these compounds because these compounds have been detected in aquatic environments and they have been found to bioaccumulate in aquatic organisms.

As this report went to press, a recent study described adverse effects of 2,3,7,8-tetraCDD and 2,3,7,8-tetraCDF at even lower concentrations than previously reported (Mehrle et al., 1988).

This chronic study of rainbow trout was a 56-day flow-through experiment consisting of 28 days of exposure followed by 28 days of depuration. At 38 parts per quadrillion 2,3,7,8-tetraCDD, the lowest concentration tested, significant effects were observed on growth and survival. Because effects were observed at the lowest level tested, a no observed effect concentration (NOEC) could not be derived.

The CDF also was very toxic. At 0.9 parts per trillion (ppt) 2,3,7,8-tetraCDF, growth was adversely affected; survival was reduced at 4 ppt. NOEC values were 0.4 ppt for growth and 1.8 ppt for survival. During the 28-day depuration period, mortality continued and there was "no apparent recovery in clean water" in both the CDD and the CDF experiments. In addition to survival and growth, the authors monitored five behavioral changes: reduced feeding, lethargic activity, unresponsiveness, resting on the bottom, and head-up swimming.

The same study determined bioconcentration factors of 39,000 for 2,3,7,8-tetraCDD and 6,049 for 2,3,7,8-tetraCDF, factors higher than previously described. Mehrle et al. (1988) concluded that 2,3,7,8-tetraCDD is more than 10,000 times as toxic to fish as the insecticides endrin or toxaphene and that 2,3,7,8-tetraCDF is roughly 1,000 times as toxic.